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INDEX OF MEDICINE





INDEX
OF
M E D I C I N E

A MANUAL
FOR THE USE OF SENIOR STUDENTS
AND OTHERS

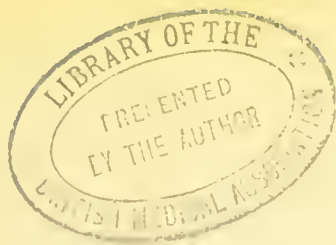
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TO

WILLIAM MILLER ORD, M.D., F.R.C.P.

WHO HAS ALWAYS BEEN MY FRIEND





PREFACE

THIS work was commenced with the view to its being a companion to Keetley's 'Index of Surgery.' It is, however, an index in its title only. It was found that the nomenclature of medical diseases, if taken alphabetically, would entail so many and such complex cross-references, that the work would be disjointed and useless. From many inquiries I find that the student does not like to meet such a reference as 'Asthma, see Diseases of the Bronchial Tubes,' but prefers to read the Diseases of the Respiratory System under one heading.

My chief object, however, was to compile a work which should be a handy manual for students preparing for their final examinations in medicine at the various examining Boards. The book has been written from notes which I have taken from the systematic and clinical lectures delivered by Doctors Peacock, Murchison, Bristowe, Ord and Smith-Shand; supplemented by references, in many instances, to standard works on pathology and medicine by various leaders in the profession.

The book, therefore, can in no sense be considered as a text-book ; but it is hoped that students may find it a useful supplement to the treatises on medicine which have been written by Bristowe, Fagge, Frederick Taylor, Roberts, Osler, and others.

A description of Diseases of the Skin has not been written. I feel that broader information would be obtained from special works devoted to this subject.

No one can be more conscious of the many imperfections of this manual than I am. But I ask the indulgence of students for whom it has been chiefly written.

I have, in conclusion, to express my thanks to my colleague Dr. W. ALDREN TURNER, for much valuable criticism and suggestion ; and also to my friend and fellow-student Mr. PERCY POTTER, F.R.C.S., to Mr. W. ORME, and to Dr. WILKS and Dr. A. ELLIOT, late House-Physicians to the West London Hospital, who have carefully revised the proofs.

SEYMOUR TAYLOR.

16 SEYMOUR STREET, PORTMAN SQUARE, W.
June 1894.

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INDEX OF MEDICINE



INTRODUCTION



DISEASE

Definition.—Any deviation from the healthy standard, either in structure or function.

It is not necessary, in considering the question of disease, that we should be able to see it, or to watch its progress and the methods by which it works. The effects which are produced are only the results of the progress of disease, and are called symptoms, or signs. Yet, in many instances, these symptoms are dignified by being called '*Disease*,' whilst the original disturbance of function, or modification of structure may not be known, or misconceived. And, on the whole, this is not only convenient, but in many cases it is descriptive. We are enabled thus to identify certain maladies which are difficult to define, and we also give to a disease a name which is derived from perhaps its most important symptom. To be exact, however, we must not look upon a poisonous material or foreign body introduced into our systems as a disease. It may remain there for an indefinite period, and produce no deviation from a healthy standard either in function or in structure. It is only when such poison acts as a morbid matter, or produces definite histological changes, that disease can be said to be constituted.

Nor is it necessary in this small work to define what *Health* is. We may easily affirm that it is a state of body and mind which is perfect in structure and in function. Even this statement, however, we are probably unable to grasp. Possibly every person on this globe is the subject of disease more or less severe. Hence the difficulty which arises in using the word 'health' as a standard of comparison. Diseases may exist of which the sufferer has no real appreciation, on account of his ignorance of true health, and they may be so trivial as to cause no symptoms by which they can be recognised. It is only when events are produced which are recognised by the sufferer, or detected by medical science, that we can really say, 'This is disease.' And such a condition may vary from slight disturbances—none the less diseases—called *ailments* or *disorders*, to the gravest states in which the body perishes in a few hours.

Alteration in structure is known as Morbid Anatomy, whilst the study of Pathology is based upon alteration or perversion of function. In some diseases, only one of these conditions exists; in others there is a combination of both. Again, alteration in function may cause change in structure, and *vice versa*.

Ætiology, or a study of the causes of diseases, is a wide subject, inasmuch as it has important bearings on prevention and treatment. Not only must the germs of disease exist, but they must fall on a suitable soil or nidus in order to flourish and produce their fruit. In a given population, whose members are all equally exposed, some will contract disease, others will escape. This fact points to certain conditions, such as age, occupation, surroundings, which act as predisposing factors in producing disease. Hence, in medical works generally, the causes of disease are divided into two large classes, (*a*) *predisposing* and (*b*) *exciting*.

For example, a young man, exhausted and perspiring, after a severe struggle or exertion, lies down on the damp grass, gets chilled, and in a short time afterwards develops fever and all the other symptoms of pneumonia. Here the exhaustion and perspiration act as predisposing causes, and the chill,

or, possibly, some specific poison, as the exciting cause. It is necessary to give some details of these two groups.

(a) PREDISPOSING CAUSES.—1. *Age* is most important. As a rule, all specific fevers are contracted more readily by children than by adults ; or, at least, these diseases are more frequently met with in childhood. The former statement is probably correct, since diseases due to specific germs appear to run a truer and more typical course—being more perfect in eruption, duration, and decline—in children than in adults. It would seem as if the very febrile organisms themselves revelled in the healthy young tissues which are so vigorous at the periods of the active development of the body. Another factor, however, to be remembered is that adults are rendered immune—at least, so far as the commoner exanthemata are concerned—by having contracted these diseases during childhood.

But there are exceptions to the above rule. It is notorious that in some epidemics of scarlet fever or diphtheria, the infant at the breast escapes, whilst all the other inmates of the house may suffer. Again, typhus fever is much more likely to be acquired by a nurse who is over thirty years of age than by a younger one.

Amongst the many diseases upon which the age of the patient has an important bearing, we may generally affirm that youth is a period prone to contract acute catarrhal disease, and that age is associated with diseases due to degenerative structural changes.

2. *Sex*.—On this point it may be asserted, as a general rule, that functional disorders are more frequent in females, and that structural diseases occur oftener amongst men. We must bear in mind the various disorders which attack girls at puberty and shortly afterwards, such as gastric ulcer, obstinate constipation, and the like. From these conditions boys seem almost exempt. We must also consider the mental and other nervous disorders which are apt to supervene at the climacteric period. On the other hand, men, by their occupations, exposures to changes of climate, and their generally harder, and perhaps more dissipated lives, are attacked by

certain diseases, such as locomotor ataxia, gout, syphilis, in greater proportion than women. But it may be stated, without fear of contradiction, that women's complaints are less severe—or, rather, that they bring under our notice disorders of a more trivial stamp than men do. This applies to consulting-room as well as to hospital practice.

3. *Heredity*.—The great tendency in Nature's operations is to produce and perpetuate healthy life; disease once acquired has a tendency to become less marked in the next generation, and finally to die out in succeeding ones. If this were not so, who would escape? Who of us could go back two or three generations without discovering a gouty, or a tuberculous, a cancerous, or a syphilitic ancestor? And, after all, this is really a proof of heredity in certain diseases, and is not an argument against it. Life tends to improvement and perfection; but influences of parentage may for the time be so strong as to overcome the reparative efforts of Nature, and so afford, in the next generation or generations, actual evidences of, or tendencies to the acquisition of, certain ailments. Thus, the child of phthisical parents presents a weak chest, and will, in all probability, itself become consumptive. Gout, again, is found in the son of a gouty father. If one of the parents, however, is healthy, the chances of hereditary transmission of disease is lessened, the poison, as it were, being diluted.

Amongst hereditary maladies may be mentioned epilepsy, and other neuropathic conditions, tuberculosis, cancer, gout, syphilis and diabetes.

4. *Climate and Locality*.—The influence of climate in causing disease is well known. The extremes of heat and cold may each be represented by diseases peculiar to them. Thus, cholera is essentially a disease of the tropics; frost-bite occurs in the arctic zone. Damp localities and damp undrained soils are noted for the prevalence of tuberculosis, bronchitis, and other diseases which attack the respiratory tract. We may, speaking in a general way, state that the liver is more likely to become affected in hot climates, whilst the lungs are spared from disease; in cold climates the reverse obtains.

As regards locality, we note the prevalence of rheumatism and cardiac disease in Derbyshire ; of goitre in certain Swiss valleys ; of vesical calculus on the east coast of England and Scotland. Disease of the blood-vessels is common in high altitudes. Leprosy is most prevalent at the sea level. We cannot, with our present knowledge, always say why certain diseases prevail in certain localities ; but we know the tendencies, and we see the beneficial results which accrue from removal from such places.

5. *Occupation*.—Certain occupations lead to certain diseases. For example, lead workers are predisposed to lead poisoning. A better example, perhaps, would be to say that they are predisposed to acquire gout. The occupation of a clerk tends to produce phthisis ; a soldier or a sailor is liable by his calling to develop aneurysm ; a worker at textile fabrics is prone to become bronchitic. Similar examples are numerous.

6. *Social Conditions* as predisposing agents are of great importance. There are diseases incidental to wealth, whilst others follow the footsteps of poverty. Typhus is never seen amongst the better classes, except in parsons and medical men ; gout is almost the exclusive property of the affluent. Punch's aristocrat was nearly correct when he said to his gouty dependent, 'I did not know that people in your station suffered from gout.' The dietary of the poor renders them prone to tuberculosis, also to intestinal worms and other parasitic diseases.

Those are best able to survive an illness, and resist the onset of disease, who enjoy a generous dietary.

Further, the poor have not the means nor the opportunities to ward off impending disease, nor can they combat hereditary tendencies to any special disorder. The rich, however strong hereditary taint or family tendency to disease may be, do not necessarily become victims ; the disease is kept at a distance by changes of air, good food, and cleanliness.

7. *Temperament*.—Perhaps more importance has been assigned to the so-called bodily temperament in the production of disease than facts justify. Nevertheless, in communities

there are peculiarities of temperament which render certain individuals prone to contract disease. This peculiarity may be overridden by other influences, or diverted into other channels ; and hence there is a tendency to acquire other special diseases, independent of temperament. This is called *Idiosyncrasy*. But from the earliest records of Medicine, temperament has been regarded as an important factor. Four varieties at least have been described, viz. the *Sanguine*, the *Nervous*, the *Lymphatic*, and the *Bilious*.

The Sanguine man, with large limbs, a full artery, impulsive movements and quick thoughts, is supposed to be prone to acute inflammatory affections, often of considerable extent, which run a rapid course to a favourable termination.

The man of Nervous temperament is of higher intellectual stamp. With small muscles and bones, a large skull, an anxious and careworn face, he not infrequently goes through severe bodily strain by reason of his extra nerve energy. The hands are lank, bony, and mapped out by full veins : their very expression of deftness is an index to the mental activity. Such people develop epilepsy and other neuropathic conditions ; they are also prone to congestions and hemorrhages.

The Lymphatic temperament is denoted by pallor of complexion and of skin generally, due to a deficiency of red, and an excess of white corpuscles in the blood. The subjects in this class are liable to dropsies, splenic diseases and chronic ailments generally. Their bodily development is coarse and flabby ; their mental powers are noted for their firmness and plodding persistency.

The Bilious temperament is characterised by mental despondency and bodily inactivity. Members of this class are liable to disturbances of the hepatic and digestive functions.

Of late years the consideration of temperament in the causation of disease has lapsed into neglect, and as a guide to diagnosis it has been overshadowed by our improved means of investigation. Further, probably no patient is typically a representative of any particular class of temperament ; and he is not illustrative of a given disease, but presents a

study of the individual. Each case, therefore, is, as it were, a new book to study. There are in Medicine no cut-and-dried precedents.

8. *Mental Conditions*.—Excessive mental strain predisposes to disease. Gout occurs in the overworked politician ; typhus fever is more likely to attack the defeated and depressed army in greater proportion than its victorious enemy ; parturition is of more serious moment in the highly strung neurotic woman than in her more composed and equable tempered sister. Numerous other examples could be cited.

9. *Previous Diseases*.—There is no more important subject as bearing on predisposition to further disease than previous ailments. When we study the records of hospital cases we are at once struck by the importance which is attached to a history of alcoholism in the causation of liver diseases, of syphilis in lesions of the nervous system, and of rheumatism in cases of heart disease. Other examples are afforded in ague, which predisposes to leucocythæmia, in chronic suppurative diseases, which tend to cause pyæmia, and also lardaceous degeneration.

10. *Bodily Strength* is also an important factor. Medical advisers are constantly being asked whether it is advisable or necessary to remove this or that person from any house or locality. And the question is important, inasmuch as epidemic diseases show a tendency during the early part of their occurrence, to select as victims those who are of poor physical strength or in low bodily health. Nor is this observation limited to epidemic diseases. It is an incontestable fact, both in the vegetable and animal kingdoms, that disease attacks by preference, and is more virulent in, those plants and animals which are of sickly growth and poor vigour. Not only are such individuals less able to withstand physical trials, which do not affect more robust specimens, but they appear prone to contract disease which passes over their more vigorous companions.

(b) THE EXCITING CAUSES of disease are equally numerous and complex in their behaviour. And in many instances it is difficult to estimate the exact part which is played by many

conditions which directly cause disease. In some instances, such as excessive eating and drinking, undue mental and bodily strain, both predisposing and exciting causes of subsequent disease, are found in the same abuses. We have, however, endeavoured to include in the following group all the principal exciting factors ; but the list is by no means complete.

1. *Mechanical Causes*.—This heading will include all the diseases of bones, joints and organs which are directly due to *blows* and *injuries* of whatsoever kind. Examples are afforded by acute peritonitis following on a kick or blow on the abdomen ; by meningitis secondary to injury ; and by an attack of acute gout supervening on wearing a tight boot. Similarly, *excessive muscular strain* may start herniæ in some subjects, or it may be the direct cause of lateral sclerosis in another patient, whilst laryngitis, caused by constant shouting, may be found in a third case. The action of *heat* and *cold* on the respiratory, cutaneous and digestive systems, again, produces inflammatory disorders of these respective parts of our system, purely in many instances by mechanical agency.

2. *Chemical Poisons*.—It is only necessary here to mention that well-known dire effects are produced, not only on the stomach and other organs, but upon the system generally, by poisons introduced from without. Many of the arts and manufactures are carried on in atmospheric and other surroundings which are absolutely hurtful to life. Painter's colic, as a result of lead-poisoning ; salivation produced by working with mercury ; and necrosis of jaw, frequent in phosphorus-match makers, are examples which occur to us at once. And in the same category must be included the various diseases which are produced by excesses in food or in drink. Obesity is a disease due, in many instances, to over-feeding. A form of nervous dyspepsia is common in tea-drinkers. The results of constant alcohol drinking are evident to us, at every visit to the out-patient room.

There is another class of poisons which is none the less a potent factor in producing disease, although not directly

coming under the notice of the toxicologists. We refer to those toxalbumins and other poisons which are generated by the arrested functions of the skin, kidneys, bowels, lungs, liver, and other organs. These poisons may be known as *Autogenetic*, and are amongst the most powerful agencies in the direct causation of grave diseases.

3. *Malformations and Malpositions*.—It is a question whether malformations, so long as they do not interfere with the healthy functions of life, would really come under the category of disease. For example, cases of complete transposition of viscera have been discovered by accident in people who suffered neither distress nor inconvenience. Supernumerary digits, again, have been observed in people who lead active lives and who live to advanced ages. But, on the other hand, malformations may directly threaten life, owing to some important organs being implicated. For example, abnormal arrangements of peritoneum may directly cause hernia, or incarceration of bowel ; a dermoid cyst may develop in relation with the abdominal cavity or elsewhere, and directly threaten life. It is only necessary further to mention malformations and arrests of development of the heart, and other vital organs, which are direct causes of diseases and conditions usually sooner or later fatal.

4. *Malignancy*.—We have put this heading under exciting causes, not so much on account of our knowledge of the subject as of the uncertain position in which malignant neoplasms stand at the present time. We can readily understand the dissemination of malignant growths throughout the body when once a malignant tumour has formed. Such secondary growths, as they are termed, easily find channels for propagation by the blood, lymph, and perhaps other media. But the cause of primary malignancy is not so evident. Setting aside the possible factor of constant irritation of tissues as a cause of, say cancer, in which case the irritation is the exciting cause, it is obvious that many malignant growths primarily occur in situations where no such agency as injury or irritation could have existed. Again, in certain localities and amongst certain communities 'malignancy' is a common condition. The

inhabitants of such localities appear prone to be the victims, not only of tumours, but of tumours which eventually reveal their malignant character.

It would suggest to us that there is some condition of body or constitution, as yet not understood, in which an ulceration, say of the uterus, shall become malignant in one individual, and was probably malignant from the first, whilst a similar lesion in another individual shall have benign characters and quickly heal under treatment. A certain morbid condition, or *Dyscrasia*, exists, which may be an exciting cause of disease.

A similar line of argument applies with even greater force in sarcomatous tumours. There must be some peculiarity, whether of temperament, or what is vaguely termed 'constitution,' exciting to malignancy certain growths which, to all appearances, should have remained permanently benign. We state this opinion notwithstanding the tendency of recent pathological research to attribute the origin of certain malignant growths to the influence of parasitic invasion.

5. *Specific Organisms*.—Bacteriological study has of late years opened out a field of inquiry as to the causation of diseases which was hardly dreamt of by earlier investigators. The tendency, however, and with just grounds, is to attribute nearly all, if not all, specific diseases to the agency of special parasitic invasion of the blood and tissues. These parasites belong to the vegetable or to the animal kingdom, the former being the most potent, most widely diffused, and most injurious in their actions. There can be now no doubt that tuberculosis cannot exist without the agency of the bacillus tuberculosis; that enteric fever and cholera are due to and caused by infection or inoculation, probably at the bowel, of specific germs peculiar to each; that pyæmia and the various forms of septicæmia originate in similar methods.

Numerous other examples could be quoted, but it is enough to say that each variety of poisonous microbe is the direct cause of a disease, and that by whomsoever these germs are received, under favourable circumstances, certain well-defined events and symptoms supervene, run a definite course,

and then subside, exactly in the same way that a vegetable seed, when placed in the earth, germinates, produces flowers and fruit, and eventually fades.

In respect of animal parasites as direct exciting causes of disease, it will be only necessary to instance the cystic stages of tapeworms, the invasion of our muscular systems by *trichina spiralis*, and of our cutaneous system by lice, itch mites, and other lowly organised members of the animal kingdom.

Contagion and Infection.—As having most important bearings on the causation of disease, it is necessary to say a few words on these subjects.

By **CONTAGION** is meant the communication of disease from A to B by direct contact. Vaccinia, syphilis, surgical erysipelas, and glanders are well-known examples.

The course of a disease which is thus acquired runs through certain definite stages. At the point of inoculation nothing is seen for periods varying in length from a few hours to many days, or it may be months. Indeed, it often occurs that the point of entrance of the disease is never discovered, and not even suspected, until acute symptoms direct our attention to the investigation of all possible sites of intrusion.

This stage of quiescence is known as the *incubative period*. Subsequently, at the point of inoculation, there appears a typical lesion, whether it be papule, pustule, or what not. From this point the contagious matter extends to, and is received by, neighbouring lymphatic glands, which again disseminate the poison, and probably in some method cultivate or elaborate it. As the poison circulates in the blood, it is increased in a ratio which we are unable to estimate, but eventually it perishes or loses its active qualities by impoverishing or altering the very fluid or tissue on which it flourished. The body of the patient is then immune, and his tissues are sterilised, so far as the specific poison under consideration is concerned.

By **INFECTION** we understand that a disease may be conveyed from A to B by some intermediate agent, such as by the atmosphere (as in influenza epidemics), by the clothes (scarlet fever), or by food (diphtheria).

Contagious and infectious diseases are therefore due to the growth in the living body of specific living organisms, mostly of low vegetable type. These may be preserved for an almost indefinite period by dirt and filth, by clothes, or in hermetically sealed tubes and vessels. But with few, and possibly no exceptions, there must have existed the initial poison from which subsequent attacks developed. If this doctrine be correct, it follows that no specific disease can originate *de novo*. Murchison, however, was distinctly of opinion that typhus could arise from the concentrated exhalations of people crowded together, and without the assistance of a previous case of the fever.

Semeiology.—Some account of the signs and symptoms of disease is important. 'Symptoms' include the greater evidence of disease, and the word is therefore a wider term than 'signs.' There are many symptoms of disease which are also signs; but, on the other hand, there may be signs, such as age, sex, and temperament, which are not symptoms.

SYMPTOMS may be defined as those evidences of a deviation from normal health which are known to the patient himself, and betray themselves to the medical man. Symptoms therefore are subjective, and are related to us by the patient, or they are phenomena which are evident to an observer.

SIGNS, on the other hand, are objective. They are recognised by our sense of touch and hearing; their intensity is increased by the aid of instrumental investigations. They are equally as important as symptoms; but at times the weight of the one set of evidence is greater than the other.

As an example of the differences between these two groups, signs describe, as it were, the physical condition of a heart or lung, whilst symptoms denote the functional derangements which such conditions give rise to.

It is obvious that signs are more important than symptoms in investigating disease of the lower animals, or when examining an unconscious human being. On the other hand, symptoms as related by an intelligent patient are in the majority of instances of the utmost value.

The symptoms of various diseases differ considerably in

character and type. Most diseases, however, may be grouped under three categories, so far as their typical symptoms are concerned. In the first group the symptoms run a definite and regular course, usually ending in recovery. Scarlet fever is a good example. In the second group the symptoms are progressive from bad to worse, and have always an unfavourable termination, *e.g.* cancer. In the third group the symptoms are irregular: the disease may abate in two or three days, or it may last as many weeks; frequently some of the principal symptoms are absent or abortive, and their sequence is not necessarily in definite order or severity. These diseases have therefore no necessary course, duration, or favourable termination; relapses are not infrequent, and their character and duration are much influenced by age, sex, occupation, temperament and general surroundings. Acute rheumatism is a good illustration.

The symptoms marking the termination of disease also vary considerably; but they are conveniently described under two headings, viz. *Crisis* and *Lysis*.

In diseases which terminate by *Crisis* there is generally at some known date a turning point at which the disease either becomes favourable or unfavourable. The term is, however, generally used to imply a favourable change in the symptoms, but not necessarily always so. Critical symptoms which are most usually observed are diminution of the pulse-rate, fall, or occasionally a rise, in the temperature to near the normal level, and some other well-marked symptoms, such as delirium, or sweat, or diarrhœa, or a copious excretion of urine. The duration of the crisis varies. In some cases it lasts a few hours only, but is rarely longer than one to two days. If the patient survive this period, he is to all outward appearances well again, although necessarily exhausted. On the other hand, a crisis may represent the sudden storm which ends in disaster.

In other diseases which terminate by *Lysis*, there is also at some indefinite period an improvement in pulse, temperature, condition of tongue, and character of secretions; but here the amelioration is gradual, recovery is slow and protracted, and it

may even be impeded or arrested by various complications, which again lead to other chronic diseases and possibly to death.

DEATH may be due to general exhaustion of the system, the vital power becoming extinguished ; or it may be caused by cessation or failure of one or more of the necessary or important functions of the body. It may occur with great suddenness, as in rupture of an aortic aneurysm ; but in the great majority of cases it ensues gradually, the body clinging to life as it were, and only relinquishing its hold after a prolonged struggle, or 'death agony.' In this state, besides the ordinary symptoms of the disease from which the patient is suffering, there are superadded those which indicate failure of the muscular and the nervous systems. The heart's contractions are irregular, the respirations become slower, bronchial secretions accumulate in the larger tubes ('death rattle'), the lips are dry, the œsophageal reflex is abolished, so that deglutition is impossible, the conjunctivæ are insensible, and the face becomes pale or livid, and is usually bathed with a cold and clammy perspiration.

The temperature in some diseases falls at this period ; in others, *e.g.* typhus, it rises, and may continue to rise for some hours after death has supervened.

It is common with most writers to attribute the cause of death to failure of the functions of the *heart, lungs, or brain*. But it is more than difficult in the great majority of fatal cases to say how far this is true. It is probable in cases in which death is said to begin at the heart, that the lungs have had an equal share in the immediately fatal process ; or, again, that the nervous system is in no small degree responsible for death which is said to originate in the lungs. It may, however, perhaps be stated that heart failure (syncope) and arrest of respiratory functions (asphyxia) are indicated in most of the symptoms in which death supervenes either suddenly or gradually.

Syncope may be induced either by sudden loss of the muscular power of the heart (asthenia), which thus becomes distended in all its cavities by the accumulated blood, or it

may occur from want of blood itself. This may be brought about by sudden loss, as in copious hæmorrhages after parturition ; or it may be caused by want of proper food, mal-assimilation and the like, as in anæmia. Here the heart cavities are empty, but always irritably contractile. In both, the symptoms are similar, except that in asthenia the mental condition remains clear and unimpaired almost to the last moment ; whilst in anæmia, convulsions and delirium are common.

Asphyxia is caused by arrest of the respiratory function. The blood does not circulate through the lung capillaries and is not therefore oxygenated. This condition may be caused by paralysis of the diaphragm and other respiratory muscles, by admission of air to the pleural cavity (pneumothorax), also by obstruction to the entrance of air to the respiratory tract, as in diphtheria, croup, and drowning. After death the left cavities of the heart are found empty, whilst those of the right side are engorged, a similar condition being found in the cavæ, liver, spleen, and other organs.

Another form of death is said to commence at the brain (*Coma*) ; but, except in severe injuries to the brain and medulla, it is probable that diseases involving the nervous system usually lead to death by implication of the respiratory or circulatory functions, since, although in coma the nervous system is primarily involved, the stupor may be so profound as to prohibit the reception of centripetal and the arrest of centrifugal impulses, and hence motor power is arrested and the blood no longer aerated ; death therefore appearing to be due to respiratory and circulatory failure.

The above classification of the causes of death is arbitrary, and, as we have previously hinted, many causes of death have no special obvious seat in either brain, heart, or lungs singly, but are dependent on two or more processes. Each system of our bodies seems to play its part in producing the various modes of dying, and they react on each other so as to prove that the body is not so much a series of automatic mechanisms as one complete whole of which the various organs are component and necessary parts.

Diagnosis tells us what the disease is. It is a study of the first importance in medicine, since treatment without diagnosis, although frequently successful, may be injurious, and is often hazardous.

Some diseases are so obvious that the least power of observation detects their characters ; but even in such instances the diagnosis of possible complications is of great necessity. Other diseases are more obscure, the difficulty in diagnosis arising not so much from lack of symptoms, as from their resemblance to, or to the occurrence of similar, signs in other disorders. In such cases we arrive at a conclusion by the processes of comparison and exclusion. This method is especially necessary in diseases of the nervous system. Some signs and symptoms are more obvious and conclusive than others. They are called *diagnostic* signs. Others, again, are so much more conclusive as to be unmistakable in their import, and are practically unknown in any other condition. They are called *pathognomonic* signs.

In forming a complete diagnosis we must inquire into all controlling signs, such as age, sex, temperament, and habits ; all possible causes, such as occupation, previous illnesses, and exposures to infection ; and then we should proceed to the investigation of all and each of the symptoms, and to the examination of all organs from which such symptoms could possibly originate.

Even then it frequently occurs that an assured diagnosis cannot be made, and it often happens, in diseases which are suspected but not confirmed, that treatment will aid us in diagnosis.

Prognosis is the art by which we foretell the course, duration, progress, and termination of a disease.

The prospect of recovery, the length and character of the ailment, and the question of suffering or distress during its progress, are matters of the first importance to patient and friends alike. In all cases our prognosis should be probable only, and although some diseases tend to recovery, and others to end fatally, any certain prediction may be falsified on account of many modifying agencies which

may come into operation. The apparently hopeless case often recovers and the most trivial ailments occasionally prove fatal. Medicine can never be regarded as an exact science.

Some diseases run a definite course, and we may safely say, unless death occur before, that the disease should have spent itself at a given date. Such an example is afforded by typhus fever, which ends abruptly on the 14th day. Other diseases run a fairly regular course, their date of termination being within a narrow limit of two or three days. Pneumonia is an instance of this. We expect to see the febrile process abate between the 5th and 8th days. Yet other ailments have no regular or definite course. In some instances, recovery soon occurs, whilst in the next individual it is delayed or protracted for no obvious reason. Chorea is a type of such cases. Therefore, in forming a prognosis, we must not be guided entirely by the general symptoms and natural history of a disease, but we must be influenced by the disease as it appears in, and is modified by, the individual. Thus, the controlling bias of age, habits, occupation, and previous illness, all bear important parts in the chances of recovery. Nor must we forget that in one person any disease may be mild in character, whilst in the next case, with identical surroundings, it assumes a grave type.

It will be seen, in the following descriptions of various complaints, that certain signs, such as general dropsy, picking at the bed-clothes, aphthæ in the mouth, are all regarded as of unfavourable omen ; but not necessarily so. We are seldom warranted in giving an opinion which destroys all hope in the minds of patient and friends ; nor, on the other hand, are we justified in lightly giving false encouragement in cases which our experience tells us generally terminate in death.

TREATMENT constitutes the various methods by which we (a) prevent, (b) relieve, or (c) cure disease.

(a) *Preventive* or *prophylactic* treatment is an important subject which is guided not only by the family medical adviser, but also by the State. It is essential in all zymotic

and contagious diseases, and then consists practically in following hygienic laws as regards food, drink, occupation, cleanliness, exposure to infection and the like.

In certain diseases, however, it has another important bearing, inasmuch as we are concerned in obviating the occurrences of complications and sequelæ in diseases, which may be more dangerous than the primary ailment itself.

(b) *Palliative* treatment consists of the various methods by which we soothe mental anxieties, relieve bodily pain and prolong life. The term is usually associated with hopeless diseases, but by no means invariably so. We relieve the pain of an inflamed joint by hot fomentations; we arrest the anguish of biliary colic by opium; and it is the proud privilege of our profession to frequently allay mental distress by our adoption of the sacred position of a father confessor.

(c) *Curative* treatment comprises the various methods, therapeutical, dietetic, climatic, mechanical and the like, by which we hope to arrest, cut short, or actually counteract the effects of disease, and so restore the body to health. It includes many different methods of cure, some totally opposed to others. The principal subvarieties of curative treatment are known as (i) the Expectant, (ii.) the Stimulant, (iii) the Antiphlogistic, and (iv) the Therapeutic. But here, again, no sharp definition can be made. Certain methods of stimulant treatment may almost be regarded as therapeutical, and also antiphlogistic treatment occasionally trenches on expectant principles.

(i) *Expectant* treatment is mainly employed in obscure diseases. We do nothing for the patient except perhaps keep him in bed, or confined to the house, or restrict him as to his dietary. In other words, we wait on Nature to reveal the character of the disease, or, that already being afforded, to effect a cure. The theory of this variety of treatment is based on the fact that many diseases tend to spontaneous cure, or at least to remain stationary, or to be only slowly progressive in spite of all our skill. It assumes that the body clings tenaciously to life, and that death only conquers with difficulty.

(ii) *Stimulant* treatment comprises the method of cure by remedies which increase the vigour and vitality of the patient. It is employed in cases in which the various functions are in a state of low activity and the bodily strength impaired. Alcohol, highly nourishing food, certain drugs, such as ammonia, are our chief agents; but we are of opinion that in many instances the so-called *sun* cures, *water* cures, and *climate* cures act chiefly through the exhilarating effects which they produce on the mind, the body generally, and also on the various secreting organs.

(iii.) *Antiphlogistic* treatment is generally known to the public as 'lowering' treatment. There is no subject in medicine about which the vulgar talk with so much confidence as this method of treatment. It probably is a survival of former days, when every patient was bled, no matter what his ailment, when blistering was used over every internal inflammation, and when every fever was treated by a dietary next door to starvation.

Bleeding is nevertheless a valuable remedy in suitable cases; a course of purgatives in certain diseases is essential; a seton or a moxa may each in its turn help in effecting a cure; diaphoresis is one of the best methods of curing a pleural effusion; and a low dietary has been ably advocated by eminent authorities in the treatment of otherwise inaccessible aneurysm. In most instances this form of treatment is eliminative in its object.

(iv) *Therapeutic* treatment is the most important of all. Every physician nowadays writes a prescription; and even those who apparently most decry the use of drugs (a tendency which is easily acquired by the younger practitioner) probably prescribe drugs more frequently than they themselves imagine. The faith of the public in the use of drugs is unbounded, and in hospital practice, at least, our patients attribute much more value to nauseous draughts than to advice as to mode of living.

The drugs which are of service in actually curing medical diseases are few; they can almost be counted on one's fingers. Arsenic and quinine cure *ague*; mercury and the iodides cure

syphilis ; ipecacuanha is almost a specific in dysentery ; but few other medicaments are actually powerful to arrest the great number of ailments. Nevertheless, the constituents of our pharmacopœia are useful in curing diseases, although some may act through the imagination of our patients ; and such aid has no right to be sneered or scoffed at. Further, certain remedies appear to cure or relieve we know not how. Our experience tells us that this drug or that combination of drugs apparently cures, or hastens the cure of, our patient ; but as to how these results are brought about we are in ignorance. This mode of curative treatment is known as *empiricism*, and probably most of our therapeutical science is included in this category. Yet it will probably be admitted that it is better to do too little in the way of administering therapeutical remedies than to do too much. Recovery in most curable diseases takes place when the patient is properly nursed, cared for, housed, and fed.

GENERAL PATHOLOGY

ATROPHY

Definition.—A diminution in size and in the number of histological elements of any structure, consequent on lessened nutrition.

Causation.—It may be quite a healthy, or normal condition ; for example, the shrinking of the mammary gland after lactation, the shedding of the milk teeth, the obliteration of the thymus, are all examples of a normal atrophy.

The shedding of hair and the changes in the cuticle which occur in old age, are also manifestations of atrophy. Indeed, life itself, with the changes, destructive and constructive, which occur in the various tissues, may be regarded as presenting a typical picture of atrophy.

Atrophy may also be a pathological change, when it is due to—

1. DEFICIENCY IN QUANTITY OR DETERIORATION IN QUALITY OF THE BLOOD SUPPLY. It may, therefore, occur as a result of starvation, of excessive vomiting, of diarrhoea, or of prolonged discharges. It follows the dyscrasia produced by fevers, malignant new growths, consumption and the like. It is also seen in those cases where an artery supplying an organ or structure is compressed by tumours or obliterated by embolism.

2. TO LOSS OF FUNCTION, and is, therefore, attendant on disuse. Muscles which, for any reason or other, are not employed, dwindle and atrophy, and in some cases disappear almost entirely. Similarly, glands which are normally active

shrink and become rudimentary when their secretion is no longer required. A good example is found in the atrophied sebaceous glands in a bald scalp.

3. TO INTERFERENCE IN INNERVATION.—For example, a muscle when cut off from its trophic centre atrophies independently of disuse. This is well seen in the wasting of muscles which accompanies lead palsy and other forms of paralysis.

The different histological elements of the body are, however, not all affected by atrophy in the same degree. In some tissues (bone and cartilage) the change is slow, in others (muscle) it is more rapid. The extremes of loss of substance caused by atrophy are presented by nervous tissues (least) and adipose tissues (most).

HYPERTROPHY

Definition.—An overgrowth of tissue. When referring to cell proliferation alone, the term *Hyperplasia* is ordinarily used.

Causation.—1. CONGENITAL PROCLIVITY.—Here the hypertrophy may affect the whole body, so that the individual develops into abnormal size and stature, or it may affect local parts or organs only, as the mamma, penis, some particular feature, or the hands, feet, &c.

2. COMPENSATORY.—As its name suggests, this condition may be regarded almost as a healthy one. It occurs (*a*) when a gland or a tissue grows to excess, in order to balance arrest or deficiency of growth or function elsewhere. This is seen in congenital absence of a kidney or a testis, when the companion organ is hypertrophied, and carries on the functions of both : (*b*) when tissue-growth takes place in excess in order to overcome obstruction, or to keep pace with unusual demands. It is especially seen in muscular tissues, as, for example, the compensatory cardiac hypertrophy in valvular disease, and again in the thickening of the muscular coats of the bladder in urethral obstruction.

3. BY INCREASED VASCULAR SUPPLY.—Good examples are seen in the bony outgrowths of gout and other diseases, in the

course rough hairs occasionally found on moles and nævi, and in the rapid growth of nails affected by onychia.

4. BY THE STIMULATION EXERTED BY INTERMITTENT PRESSURE.—This is observed in the formation and growth of corns and callosities of the feet and hands. The pressure, however, must be intermittent so as to allow free access of blood to the part. Constant pressure alone, will cause atrophy.

DEGENERATIONS

1. Fatty

Definition.—A degenerative change characterised by the deposit of molecular fat in tissues in which it does not normally exist.

Causation.—An excess of fat may be deposited in the tissues of the body generally, by the consumption of an amount of carbo-hydrates beyond its requirements.

In the degenerative process of disease the fat is derived from the albuminous constituents of the tissues, which break up into their chemical elements, the nitrogenous portions being carried off, leaving the fatty elements behind. This change is, therefore, marked by the appearance of an excess of urea and urates in the urine. It is, however, essential, before these pathological changes can take place, that the various tissues and cells which are affected shall have previously lost some of their vitality and energy.

Fatty degeneration may occur (1) *Locally*, or (2) *Generally*

1. LOCAL DEGENERATION may result from occlusion of a blood vessel, as in cerebral embolism, where the fat is deposited in the form of free oil or of granular corpuscles of Gluge. This change occurs, not only in those parts of nervous tissue where fatty matter is a component element, as in the myelin sheaths of nerves, but also in the grey matter, where there is, normally, no fat. It is often followed by calcareous infiltration as a secondary result.

It may also be consequent on inflammatory changes in the secreting epithelium of glands, as in Bright's disease (large pale kidney). It may affect the histological elements of

tumours, which are quick of growth and thus exhaust themselves. Finally, it may attack the tubules of a nerve which is severed from its trophic centre, as in experimental Wallerian degeneration ; or it may appear subsequent to the inflammatory changes of disease (advanced sclerosis).

2. GENERAL DEGENERATION depends on some morbid change in the blood. It is, therefore, consequent on the absorption of certain poisons, such as phosphorus, arsenic, &c., or it is a condition attendant on grave blood disorders, such as are found in acute atrophy of the liver, leucocythæmia, pernicious anæmia, and the like. The pathological changes are most marked in the muscular fibres of the heart, the tunica intima of arteries, and in epithelial structures.

Whatever be the cause of fatty degeneration, the cellular elements are the most affected. Fat accumulates in the interior of the cells, at first in separate globules, like crude mercury, which eventually coalesce in the advanced stages, until the whole cell is distended as with oil, the nucleus being hidden or destroyed.

2. Amyloid (LARDACEOUS)

Definition.—A degenerative change supervening for the most part on suppurative diseases ; in which the tissue elements are saturated with a substance allied to albumin.

Causation.—It is secondary to prolonged suppuration or discharges, by which the blood becomes robbed of its albumin and otherwise profoundly altered. It is, therefore, frequently a sequel to syphilis, caries, and other diseases of bone, chronic tuberculosis, dysentery, and other diseases in which wasting is due to suppuration. It may occur in syphilis without suppuration.

The various tissues involved are lowered in vitality by the altered conditions of the blood, which allow its albuminous constituents to enter into composition with the protoplasm of the tissue cells. It would therefore appear to resemble a form of fibrinous coagulation occurring in living tissues. The so-called amyloid deposit is a modification of albumin ; deficient in potash, but containing an excess of soda and cholesterolin.

It may affect any organ in the body, but especially involves the spleen, liver, kidneys, and intestinal canal.

Whatever part is affected by the disease, the earliest commencement is in the smaller vessels and capillaries. Thus, their coats are thickened and rendered translucent and brittle; their lumen is narrowed or even obliterated. The cells of the affected organ increase in quantity, lose their nuclei, and become granular at first, and subsequently bright and transparent. Consequently the viscus which is involved becomes enlarged, swollen, heavy, pale, and anæmic.

On section the organs are translucent and waxy, resembling somewhat the appearance of cold suet pudding or of bacon.

The 'amyloid' substance is, as previously stated, a nitrogenous compound allied to albumin. It has no relation to starch, but is somewhat like dextrin in its chemical reactions, inasmuch as it turns a dark mahogany colour when immersed in a solution of iodine. The term 'amyloid' is retained on account of its general use.

The amyloid reaction can be roughly shown by pouring over a fresh section of any organ in which the disease is present an aqueous solution of iodine and iodide of potassium (Iodine gr. x., Potassii Iod. gr. xx., Water 4 oz.), when, after a few seconds' duration, the smaller arteries and capillaries, such as the Malpighian tufts of the kidney, the vascular bodies of the spleen, and the terminations of the hepatic arteries in the liver, become stained with a dark chestnut or mahogany tint, which cannot be washed out.

For microscopic sections; use an aqueous solution of gentian violet, which produces a double stain, the amyloid changes becoming pink, and the normal tissues blue or slate colour.

3. Calcareous

Definition.—A degeneration characterised by the deposit of lime salts, from the blood, in the various tissues of the body.

Causation.—It only occurs in those parts which have been injured, or have lost their vitality; and even then the calcareous salts are not properly united with the tissues: they

are merely infiltrated through, and deposited in them. The deposits may, therefore, be regarded as foreign bodies; or, rather, any part of the organism which has lost its vitality may be looked on, to a certain extent, as dead matter, which, acting as a foreign substance, causes the lime salts contained in the blood to be deposited thereon; it is a petrification from the blood, and not a change produced by any secreting power of the tissues involved. For its complete fulfilment it is necessary that the circulation in the part should be slow.

The lime salts are deposited as granules in the intercellular matrices, or in the protoplasm of cells, and resemble somewhat, both in appearance and method of infiltration, the early stages of fatty degeneration. The granules, although crystalline in form, soon become spherical owing to their precipitation in a colloidal basis, according to the doctrine taught by Rainey and W. M. Ord that crystalline substances tend to become spherules when deposited in the presence of a colloid. There is thus a struggle and a compromise between the crystalline and the colloidal bodies. As the deposits increase, the granular condition of the tissue is lost, and its structure is more homogeneous.

The calcareous deposits may be dissolved with effervescence by the action of acetic or other acids, showing the presence of carbonates.

Calcareous degeneration, although usually an indication of destruction and waste of tissue, may nevertheless represent an effort of repair. This is seen in the invasion of trichina spiralis. Here, in the later stages, the parasite is entombed or shrouded by a lime casket due to calcareous degeneration of its capsule. Similarly, the healing of tubercular lesions is accompanied by calcareous degeneration of the inflammatory products. In extra-uterine gestation the fœtus, acting as a foreign body, is encapsuled, and to a certain extent preserved, in calcareous matter. The proliferated products of valvular inflammation may undergo also calcareous thickening.

But it is especially seen in tissues which are old or past repair, as in cartilaginous structures, and in the senile decay of arteries, which hence may become rigid, brittle tubes.

4. Pigmentary

Definition.—A degeneration characterised by the deposit of pigment in parts where it does not normally exist ; or by an excess of normal pigmentation.

Pigmentation itself is not necessarily a degeneration ; but is placed here for convenience. The amount of pigment in the tissues may vary between that extreme in which it is almost entirely absent, as in albinos, and that in which it is markedly present, as in southern races, and yet be consistent with perfect health.

Causation.—1. FROM THE BLOOD.—The colouring matter is abstracted or washed out from the corpuscles ; or it may be absorbed by amœboid leucocytes and deposited in the various tissues. The colouring matter itself is hæmatoidin, a derivative of hæmatin, which occurs in rhombic crystals of a purplish hue resembling hæmin. Hence, it results from congestions, both active and passive, as in pneumonia, which frequently leaves a permanently dusky tint of face. It is also seen in the lungs itself, in the condition known as ‘brown induration.’ It may also be secondary to extravasations and bruises, as in cancers, scurvy, typhus fever, &c. Again, in certain diseases the blood may contain free pigment, due to the destruction of its corpuscles, which is then carried to all parts of the system. Examples of this condition are seen in the discolouration of pregnancy, syphilis, ague, and leucocythæmia.

Finally, the pigment may be correctly elaborated by the blood, as in retained biliary secretions, but hindered in its elimination from the body and reabsorbed, *e.g.* in obstructive jaundice.

2. FROM AN EXCESS OF NORMAL PIGMENT.—This is especially observed in tumours which originate in pigmented structures, as the choroidal coat of the eye, and the skin. The tumours of the skin, known as ‘port-wine marks,’ and pigmented nævi, would come under this category.

3. PIGMENT DERIVED FROM WITHOUT.—Under this heading discolourations are chiefly seen in the skin, though the lymph glands may be secondarily involved. A more or less per-

manent pigmentation is thus found in tramps, uncleanly persons, and those subject to exposure to sun and winds. Certain occupations also predispose to discolourations. It is only necessary to mention colliers, dustmen, asphalt workers, and the like. It is seen as a result of the direct local action of poisons, as nitrate of silver, carmine, indian-ink (tattooing.) Also, as coming under this heading, we should mention the effects of frequent blisters.

DROPSY

Definition.—An excess of fluid in the serous cavities, or in the subcutaneous cellular tissues.

Various terms are in general use to denote the locality or the extent of the effusions.

Causation.—The primary cause of all dropsies is an effusion of blood-serum from the vessels, in excess of normal absorption. The condition may therefore be (a) LOCAL or (b) GENERAL.

(a) LOCAL DROPSY occurs when, from whatever cause, there is obstruction to the return of venous blood by the veins, or when the lymphatics are mechanically unable to absorb the fluid transuding from the capillaries, or as a result of some local inflammatory condition.

Effusions may, therefore, occur into the ventricles of the brain as in hydrocephalus, into the pleura (hydrothorax), into the pericardium (hydrops pericardii), into the peritoneum (ascites), into a joint (hydrops articuli); or it may involve a limb when the lymphatics are obstructed, as in lymphadenoma: and, finally, it is seen in gouty inflammation of the sheaths of tendons, and in inflammatory conditions of the areolar tissues of the neck in scarlet and other fevers.

(b) GENERAL DROPSY, or ANASARCA, is also due to obstruction to the return of blood to the right heart, in the majority of instances. These causes are included under the heading of general *passive* dropsy. It may, however, be secondary to some abnormal condition of the blood and capillaries, such as occurs in Bright's disease and the severer forms of erythema, when it is known as *active* general dropsy.

It is obvious that should any obstruction to the venous circulation occur—whether that obstruction take the form of disease of heart valve, or of ventricular walls, or of the lungs—the venous system behind this obstruction is loaded to repletion, and secondarily the lymphatic circulation is also impeded. Hence, there is not only an increased general exudation of serum into the interstices of the connective and other tissues, but also a diminished amount of lymphatic absorption; there are then two factors at work, viz. increased transudation from backward pressure, and diminished lymphatic absorption owing to the venous engorgement.

In the active general dropsy, such as supervenes in Bright's disease, some other agencies are brought into play. The increased tension in the arterioles would favour dropsy, notwithstanding the thickening of their coats. The serous effusion in these cases therefore occurs in those parts, such as the eyelids, where the connective tissue is destitute of fat, and thus affords the least amount of support to the distended capillaries.

The general dropsy which is seen to accompany severe urticaria and erythema is probably due to some disordered condition of the nerves supplying the smaller arterioles. It may, hence, appear as a local affection only in some instances; and in others, owing to a more widely diffused neurotic influence, the dropsy may be general.

INFLAMMATION

Definition.—Certain changes which occur in a tissue, as a result of injury or irritation; as a result of efforts to neutralise such irritation; and as a result of subsequent efforts of repair.

Causation.—1. SOME DIRECT IRRITATION TO THE PART.—The irritation may be mechanical (foreign body), chemical (moxa), thermal (actual cautery), or electrical.

2. SOME DELETERIOUS AGENT existing in the blood, as microbes, poisons, &c.

3. A LESION OR ABNORMAL CONDITION OF THE NERVOUS SYSTEM, *e.g.* division of fifth nerve, causing destructive inflammation of the eyeball ; injury to urethra, producing renal inflammation by reflected action through the vaso-motor nerves.

4. SOME DISORDERED CONDITION OF THE DIGESTIVE FUNCTION by which the tissues, ordinarily invulnerable, are unable to resist the attacks of cold or injury ; hence, catarrhs of stomach or bowels.

5. PERSONAL FACTOR.—From some hitherto unexplained reason, certain subjects are particularly liable to inflammation, although the existence of an irritant cannot be detected.

Whatever the cause be, and whatever the organ or tissue affected, the course of the pathological phenomena bears marked similarity in all cases. There is a sameness and a repetition of processes in the inflammation of all tissues.

Immediately, or soon, after the application of the stimulus, certain marked changes occur in the vessels, in the blood stream, and in the neighbouring cells of the organ or tissue. The smaller arteries dilate, the dilatation being often preceded by contraction due to the immediate effect of the stimulus ; this is followed by determination of blood (hyperæmia), the vascular current being accelerated and all the vessels overfull ; then the current is slowed and finally ceases (stasis), the stagnation being accompanied by heat.

The white blood-cells are now seen to adhere to the sides of the vessels, or to roll slowly along like corks at the edge of a sluggish stream. In the tissues surrounding the active inflammation the blood current is still active and quickened.

The next event is the exudation of the serum or fluid constituent of the blood ; this may be represented by the 'discharge' of an open wound, by catarrh of a tube or duct, by dropsy of an enclosed cavity, or by œdema of areolar tissues. The transuded serum somewhat approaches liquor sanguinis in character, and may, therefore, at times (as in pneumonia) deposit a fibrinous network in the alveoli or in the network of areolar tissue.

Contemporary with the serous exudation we also observe diapedesis, or migration of the blood cells, both red and

white, through the capillaries into the structures immediately surrounding ; and with this migration, and as though stimulated by it, the cells of the connective tissue and of the epithelial or other structures into which this migration takes place, enlarge and proliferate, the congregation of cellular elements being also augmented by the fission of the white corpuscles. At this point the inflammatory process may be arrested and its products absorbed, or, proceeding, it goes on to the formation of pus and abscess, or to local death, which may be superficial only (ulceration), or which may involve the whole tissues of the part (gangrene). In chronic inflammation the process may terminate in another method, viz. the formation of new fibrous tissue, as in cirrhosis.

If the inflammation proceed to the formation of pus, the cellular elements (migrated blood corpuscles, proliferated connective tissue cells, or epithelia) become cloudy, they lose their sharp outline and nucleus, and eventually become granular or even fatty. Thus, pus may be formed from migrated blood cells, red and white ; from proliferated cells of connective tissue ; and from the proliferated epithelia of the gland, tube, or organ which is involved. Pus, unless liberated artificially, or unless it find some outlet, may remain quiescent, or, its fluid parts becoming absorbed, it remains as a thick, gummy material infiltrated with calcareous deposit.

During the height of the inflammation there is heat in the part, and fever, the latter being due to the circulation in the blood either of the abnormal elements (poisons) which caused the inflammation, or of the local products of the inflammation.

We may here briefly recapitulate the events which occur in inflammation, viz. (1) Dilatation of capillaries. (2) Determination of blood to the part. (3) Stasis. (4) Exudation of fibrinous serum. (5) Migration of blood corpuscles. (6) Proliferation of blood cells, epithelium, and connective tissue cells. (7) Degeneration of proliferated cells and formation of pus.

NECROSIS (GANGRENE)

Definition.—A localised or circumscribed death of any tissue or part of the body.

Causation.—1. BY DIRECT ACTION OF AN IRRITANT, or other agent, on the tissues. This may be brought about by chemical bodies (acid or alkaline) causing a gross lesion and subsequent local death (*e.g.* blisters, moxas); or it may result from the local chemical action produced by microbes circulating in the blood. Examples of the latter cause are afforded by cancrum oris, erysipelas, diphtheria, &c. It may also be due to mechanical destruction of tissues, as in crushes and other severe injuries. It may be consequent on local pressure, as in bedsores, or from tightly-bandaged splints. Or it may follow the effects of great heat or intense cold (burns, frost-bites).

2. DEFICIENT BLOOD SUPPLY DUE TO OBSTRUCTION OF ARTERIES, whether by embolus, disease of their coats (senile atheroma), or by spasm (Raynaud's disease, ergot poisoning).

3. DEFICIENT INNERVATION, producing trophic changes in the tissues. Good examples are afforded by sloughing of the eyeball in destruction of the fifth nerve, in bedsores following a transverse myelitis, and in cystitis, also due to lesions of the spinal cord.

The extent of the gangrene and its character vary considerably according to its causation, the structure of the part involved, and its situation, whether exposed to the atmosphere or not. In tissues where there is little or no moisture, and where there has been no antecedent inflammation, the gangrene is of the 'dry' character (senile gangrene). Bullæ form on the necrosing part, which bursting, favour evaporation and dryness. The subjacent skin becomes livid, then acquires a blue or purplish tint, and eventually turns black. In another form (moist gangrene) the parts become putrid, they retain their juices, the muscles and connective tissue are softened and separated, and finally there is a sharp inflammatory line defining the living from the dead. This form is always

attended by a disgusting odour of decomposing animal matter.

The termination of the two above-mentioned varieties consists of a throwing off, or sloughing, of the part. This is cut off from the living tissues by a line of demarcation chiefly the result of inflammatory action.

In yet a third variety, although there is local death there is no decomposition, the foreign body becoming encapsuled or embalmed with calcareous salts. A good example is afforded by extra-uterine gestation, when the fœtus may remain preserved in the abdomen for years.

Other forms of necrosis have been described, such as colliquification, and caseous necrosis.

CARCINOMA (CANCER)

Definition.—A tumour of epithelial structure, originating from epithelium. The cells of the new growth, however, by their excessive growth, or from the results of pressure, have a tendency to deviate from the normal appearance of the cellular structure from which they originate.

Causation.—**AGE.**—Seldom before puberty : and usually not till after thirty years.

SEX.—Females suffer more frequently than males, the preponderance being due to the tendency of malignant growths to attack the mammæ and uterus.

HEREDITY.—Always a strongly-marked feature.

LOCALITY.—So far as England is concerned, the disease appears to be rife in Devon, Dorset, and in some manufacturing centres. It appears, from returns, to be certainly on the increase.

INJURY.—Would seem to be a strong predisposing factor. And under this heading we must include irritation. Cancers appear in the lips and tongue from irritation caused by pipe-smoking (especially those pipes which, having a small bore, produce a concentrated current of hot smoke on a given point); in the scrotums of sweeps and dustmen; in the pylorus and at the cardiac sphincter, where hard food may be

supposed to irritate and injure in its passage ; and at the os uteri after frequent parturitions or lacerations.

GENERAL CHARACTERISTICS.—All neoplasms, whether malignant or benign, are merely repetitions or aggravations of some pre-existing tissue. A growth is said to be *malignant* when it shows a tendency to spread to and invade neighbouring tissues, and when it presents no tendency towards cure, but rather to recur after removal. Malignant growths are distinctly parasitic in two ways, inasmuch as they live upon, and are nourished by, the tissues which they invade ; and secondly, the tendency of modern pathology is towards assigning the direct cause of cancers, at least, to some parasitic (protozoan) invasion.¹

In their microscopic structure all the carcinomata present certain features in common :

1. The cells of the tumour always originate from pre-existing proliferation and activity of the cellular elements of the tissue involved. This is seen in eczema of nipple preceding cancer of breast ; in irritation of tongue from smoking, predisposing to epithelioma, &c.

2. There is not usually a limiting capsule or wall to the growth ; its cells, whether originating from endothelium or from epithelium, can be seen to merge with, and be lost in, the cells of the structure which is infected.

3. There is in all varieties a well-marked stroma or basis of connective tissue, which originates either in inflammatory hypertrophy of the local connective tissues, or it is a newly-formed connective tissue proper to the tumour itself. The amount of the stroma varies considerably in different varieties.

4. Though not so vascular as the sarcomata, vessels run freely into them, being conducted into the interior of the new growth by the interlacing stroma.

5. They are freely supplied with lymphatic vessels, and hence have a tendency to spread by these vessels, the new growth involving the spaces in the tissues which are practically the open-mouthed radicles of the lymphatics. This infection is specially seen when a neighbouring lymph gland is

¹ See Galloway, *B. M. J.* Feb. 1893.

involved. It is found in its early stage to be encapsuled by new growth, the interior being, as yet, free, owing to the distribution of the afferent lymph vessel to the circumference of the gland.

6. They advance locally, either by the extension of their epithelial outgrowths which infect neighbouring cells, or by budding offshoots insinuating themselves between the surrounding tissues.

7. They tend to ulcerate, from the pressure exerted by the exuberance of their newly-growing elements.

8. When advanced, the tumours undergo certain changes. They become fatty, their cellular elements are absorbed, leaving the fibrous stroma to increase freely. Hence, the tumour shrinks and acquires a hardened, cicatrical character.

9. The disease ultimately infects the blood, producing emaciation, anæmia, and the formation of new growths in distant parts. The secondary distribution, however, is brought about in the early stages essentially by the lymphatics, as we have seen, or by the venous system, which is in immediate relation with the original seat of disease. Thus, cancerous infection may appear in the heart or lungs, carried there by the superior cava when the primary disease is in the head or neck ; the liver through the portal vein may be involved secondarily to cancer of the bowels ; or a similar infection may be conveyed by the azygos system of veins from abdomen to thorax.

10. Those localities which are usually the sites of primary cancer are rarely secondarily involved, and *vice versa*.

11. Secondary growths are, structurally, repetitions of the primary.

12. They all cause, and are frequently preceded by, wasting and cachexia. Exceptions to this rule, however, frequently occur. We have seen one or two cases of cancer which would never have been suspected from the ruddy and well-nourished appearance of the sufferers.

Varieties. I. EPITHELIOMA.—In this variety of cancer there are two different forms : in one (*a*) the growth is penetrating and extends inwards ; in the other (*b*) the extension is lateral, the growth becoming flattened or spread out. In the

penetrating form cylinders of epidermis sink into the dermis, or into the submucous tissue as the case may be, destroying its connective tissue, and by concentric pressure on their own cellular growth produce the typical congeries of cells, or 'birds' nests,' the outer whorl of cells being flattened and almost horny in character, whilst the central cells retain their plump, rounded appearance. On the surface of the growth there is a tendency to the formation of papillæ, and this feature may be exaggerated to such an extent as to cause a cauliflower excrescence, or villous tumour as in the uterus. When this is a marked feature it has by some writers been regarded as a sub-variety (villous carcinoma).

In the spreading variety there is rapid breach of surface, an ulcer forming with overhanging or everted edges, in which the typical groups of cells are found.

Both kinds readily afford, on scraping or pressure, a juice which is rich in the cellular elements of the growth. In epithelial cancer the secondary invasion of lymphatic glands is less frequent, or occurs later, than in the other varieties.

Favourite sites for epithelioma are the skin, especially where it joins a mucous membrane. It is met with in the tongue, scrotum, and prepuce.

II. MEDULLARY OR SOFT CANCER. (*Encephaloid*).—This variety is characterised by an exuberance of cells enmeshed in a fine, scanty stroma; by the existence frequently of hæmorrhages in the interior of the growth; by its tendency to ulcerate and form a fungating tumour; by its rapid growth and its extreme malignancy. The cells are not large, but flat and angular, or polygonal. It is found especially to involve mucous membranes, the ovaries and testes.

III. SCIRRHUS OR HARD CANCER.—In this variety the conditions are the reverse of the medullary form. The stroma, formed of connective tissue, is in excess, whilst the contained cells are comparatively few in number. Consequently the tumour is hard, gristly, and often dimpled. Further, the epithelial cells are prone to fatty degenerative changes, and, becoming absorbed, leave a tough stroma which rapidly increases in the later stages until, microscopically, the tumour

consists of little else, a straggling cell being found here and there only. Its blood-vessels are few, it grows slowly, and is usually attended by lancinating pains owing to its resistance, and its adhesion to skin and neighbouring structures.

The disease especially attacks the mamma, the stomach, the ovaries and testes, and also the peritoneum.

IV. COLLOID CANCER.—This form appears to combine some of the characteristics of both hard and soft cancers. In it there is an excessive new formation both of stroma and cells. The stroma in its development forms lacunæ or spaces in which the cellular elements are lodged. Subsequently the cells undergo rapid colloidal degeneration, until the loculi are filled entirely with a gummy, colloid matter or the degenerated remains of the cells only. The colloid material, by its increase, distends and distorts the alveoli, rendering their walls thin, but hard and tense, and so producing a somewhat fluctuating tumour possessing great resistance. The growth has little tendency to metastasis, but it readily spreads centrifugally: hence, when it attacks the peritoneum, it may produce symmetrical enlargement of the belly which may simulate ascites.

It is usually found in the stomach, intestines, or the peritoneum.

V. ADENOID CANCER (*Adeno-Carcinoma*).—The chief characteristic of this variety is the formation of tubes, lined with cylindrical epithelium which increases to such an extent as to choke them. All these tubules are separated by a well-marked stroma of new formation. Hence, the microscopical appearance of the growth is not unlike that of some tubular glandular structure such as the kidney or the mucous membrane of the rectum. The growth readily infiltrates neighbouring parts, and, extending to the surface, where it is exposed to friction or irritation, it soon ulcerates. Its malignancy is very marked, but it is less frequently observed than the other varieties.

Other sub-varieties have been described by pathologists, such as Melanotic Carcinoma and Mucous Carcinoma. They appear to be chiefly new growths of one of the above types, which depend for their altered microscopical appearances on

degenerative changes (pigmentary or colloidal) which may occur in them as in any other form of malignant growth.

SARCOMA

Definition.—A new growth of malignant character which originates in embryonic connective tissues ; but, unlike connective tissues, it presents an excess or preponderance of cellular elements. Sarcomata thus differ from carcinomata in their origin, but approach them in their growth.

Causation.—Nothing is definitely known as to predisposing causes, except that the disease is more prevalent in the young as compared with cancer, and that certain growths, notably papillomata, may take on a sarcomatous condition after irritation.

General Characteristics.—1. They originate where connective tissue is abundant, and frequently, therefore, spring from bone, periosteum, skin, mamma, and brain.

2. They thus vary in microscopic character according to the connective tissue from which they spring. Hence pathologists speak of fibro-sarcoma, myxo-sarcoma, chondro-sarcoma, &c.

3. The cellular elements are rich, and, as a rule, each tumour has cells of a definite shape, either round (large or small) or spindle, or huge irregularly formed (giant) cells.

4. Occasionally a sarcoma will, in different parts, exhibit cells of all four varieties.

5. They are rich in blood-vessels which run into, and are in contact with, the cells of the growth.

6. They tend to undergo pigmentary and other forms of degeneration. Such degenerations may attack the cells or the intercellular tissue, or both.

7. A growth, which is superficial, may extend to the overlying skin : it then ulcerates and forms a foul, fungoid mass, tending to soften and bleed.

8. They have a local malignancy, by spreading along the planes of connective tissue of the part.

9. They are disseminated principally by the vascular

system, but may at times spread by the lymphatics, especially if those lymphatics at the seat of origin of the growth are involved.

10. Secondary growths are repetitions of the primary one. Therefore a secondary bony growth may appear in a gland, or a cartilaginous tumour in the skin.

Varieties.—I. **ROUND-CELLED SARCOMA.**—The growth consists mainly of cells which are round in shape, sometimes tending to become oval, and of the size of red corpuscles ($\frac{1}{3300}$ in.). It is extremely vascular, the vessels having feeble embryonic walls, with occasional dilatations or aneurysms; hence there is a frequent tendency to hæmorrhages. There is a slight reticulated intercellular tissue which can be made more apparent by washing or pencilling a section of the tumour. Its growth is rapid, and as it easily insinuates itself between the tissues there is no marked line of limitation. It is the most malignant of all the sarcomata. Its most frequent origin is bone, muscle, and mammary gland.

II. **SPINDLE-CELLED SARCOMA.**—Here again the cellular element is most evident. The cells are oval or spindle-shaped, they are held together by, or contained between, little or no matrix, the cells themselves having a tendency to cohere together in groups or bundles. The tumour itself is hard and fibrous; and, apparently from the nature of its structure, it is not so malignant as the previous variety. There appears, as it were, an effort at the formation of some definite structure which is not evident in the round-celled variety. Its most frequent site is periosteum, muscle, and mammary gland.

III. **MYELOID OR GIANT-CELLED SARCOMA.**—The large cells are the prominent features in this variety. They are huge, $\frac{1}{1000}$ to $\frac{1}{2000}$ of an inch in diameter, irregular or oval in form, and present many offshoots or processes. They also contain numerous nuclei embedded in the protoplasm of their interior.

Although giant cells are most pronounced, they never exist by themselves, but are accompanied by cells of smaller size, usually spindle-shaped or oval or rounded. The tumour is of soft consistence and brownish in colour, and frequently contains small cysts. It is of slow growth; and secondary

deposits are not frequent. It usually originates in the medulla of bones, or in the periosteum, especially that of the jaws.

IV. MELANOTIC SARCOMA. — Always originates in such tissues or organs where pigment normally exists. It is thus found in the eyeball, the skin, and other parts. The cellular constituents are oval or rounded, and contain nuclei which are pigmented. Granules of pigment are also found around the nuclei, and free between the cells. The pigment itself is black or brown. Metastasis is frequent in this form. The question arises whether melanotic sarcoma is a distinct variety, or merely a degenerative change occurring in the spindle-celled or in the round-celled form. We have seen cases of spindle-celled sarcoma which ultimately became pigmented in their secondary deposits.

Other sub-varieties have been described, such as *Cylindroma*, which is characterised by a growth containing hyaline tissue in cylinders which usually coat the smaller vessels ; —probably a form of mucous degeneration ; *Alveolar Sarcoma*, in which a stroma is present, forming loculi as in cancers, but with, in addition, a delicate intercellular growth of connective tissue.

GUMMA

Definition.—A growth composed of granulation tissue, tending to undergo necrosis, and which is characteristic of tertiary syphilis.

General Characteristics.—The growth or tumour may vary in size from a mustard seed to that of an orange, or even larger. As a rule, the smaller the growths the greater the number in a given organ ; there are generally only one or two of the larger size. Each gumma is a yellowish-white or greyish nodule, soft and elastic to the touch, and embedded in connective tissue cells, by which its outline is sharply defined as with a firm capsule. Its centre may be caseous and liquefied. It has few blood-vessels, which, however, permeate the growth and are not limited to its circumference, although better developed there.

On **microscopic** examination with a low power, the centre of the growth is found to be opaque, whilst the periphery is more transparent, being composed of newly formed connective-tissue cells mixed with fibrous tissue. When examined under higher powers the cells are seen to have nuclei and resemble the white corpuscles of the blood. They are held together by delicate connective tissue which forms a fibrillar matrix. The central cells, which are the first to become caseous, after a time shrink, their nuclei disappear, and the whole cell contents are represented by fine molecular fat.

Besides caseation, other subsequent changes may take place. If the tumour be superficial, it may ulcerate, forming a ragged sloughing sore, with swollen, irregular, and inflamed walls, so that there is then a tendency to further creeping ulceration and degeneration.

On the other hand, a caseating gumma, if deeply embedded in an organ, may remain quiescent and free from further change for a considerable period ; or it may heal by absorption and the formation of connective tissue, whilst the caseating matter dries up and undergoes calcareous infiltration.

Other subsequent changes often associated with gummata are, (1) amyloid degeneration, which may come on independent of suppuration or bone disease ; (2) arterial changes, consisting of specific patchy inflammation of the coats of the vessels, especially of the internal coat, with subsequent obliteration of lumen.

A gumma may by its juices be an infecting lesion. It is a local retention of the syphilitic virus which is stimulated into renewed activity by some cause or other so as to produce a local growth. The lesion is, therefore, not symmetrical, as the poison is no longer in the blood.

Locality.—Gummata may grow from any tissue of the body. They are found in the skin, in muscles, in mucous membranes, in the liver, and in the cord or brain ; also in periosteum, dura mater, and other fibrous-tissue membranes.

TUBERCLE (TUBERCULOSIS)

Definition.—Tubercle is a new growth in the form of small nodules, or a collection of nodules, each of which is due to an irritative process produced by the presence of a specific microbe, the *Bacillus tuberculosis*. By Tuberculosis is meant the growth and development of tubercles.

Causation, (a) PREDISPOSING.

Age.—No age is exempt. Tubercular disease may exist in the infant at the breast, and in old age. It is most common, however, in early adolescence, or between puberty and twenty-five. Murchison taught that it not infrequently first shows its presence about the sixtieth year, and that one or more members of a family were attacked at about the same age.

Sex.—The cases appear equally divided between males and females, if we eliminate those cases which are induced by exposure. This factor appears to give a preponderance to males.

Climate.—It prevails most in cold and damp countries. The disease is comparatively rare in arctic and in tropical regions. Countries in which high winds prevail, causing much dust, also appear to be favourable to the disease, the dust probably acting as a vehicle conveying the bacillus; *e.g.* certain parts of Australia, Vienna, and the plains of Central Europe.

Heredity is always a well-marked feature; especially as regards tubercular disease of the lungs.

Occupation.—The mortality from tuberculous disease is high in certain trades and callings, viz, those which entail exposure to vicissitudes and climate, such as river-side labourers, cabmen, and the like. It also prevails amongst those employed in ill-ventilated and over-heated rooms (seamstresses, compositors). Butchers, from some cause not yet fully determined, also appear prone to acquire the disease.

Diseases.—It is often a sequel to exhausting diseases, notably typhoid fever, diabetes, chronic dysentery, syphilis. Also diseases of the respiratory tract favour its development (bronchitis, chronic pneumonia, &c.). In measles, of which it

is often a sequel in those patients with hereditary predisposition, it would appear to be favoured by the characteristic lesion of the bronchial tubes.

Surroundings.—As might be expected, tubercular disease is prevalent amongst the poor, and those whose food supply is defective and deficient, or whose dwellings are damp, overcrowded, and unwholesome. We may here mention, as bearing on the subject of food, that tubercle is comparatively rare amongst carnivora, but frequent in herbivora.

(b) *EXCITING CAUSE.*—The *Bacillus tuberculosis* of Koch (see Pulmonary Tubercular Phthisis, p. 361). This has been proved beyond all question or doubt by experimental and clinical research. The bacillus is a rod-shaped body, $\frac{1}{3000}$ of an inch in length (about), with somewhat blunted ends; it contains rounded spore-like bodies; it is readily stained by solutions of fuchsin or gentian violet containing free aniline; it flourishes at about 100° Fahr.; its growth is slow, but its vitality and resistance is great, so that it springs into activity very soon under suitable circumstances, even if it had remained dormant for some time previously. It may be conveyed to the system through the respiratory tract by the inhalation of bacillus-laden atmosphere, or dust impregnated with the dried sputa of tuberculous patients; by the mouth (milk or flesh of infected animals); or it may enter by the skin (lupus), or by the lymph-glands. In the latter event there has usually been some pre-existing lesion of skin or of mucous membrane.

We are thus all liable to the disease: we breathe it in or we eat it, or we are inoculated through some superficial breach of surface. But we do not all cultivate the bacillus in our tissues; besides the bacillus, a suitable nidus or medium is necessary for its growth, and this nidus is not ordinarily presented in robust, healthy subjects. In proof thereof many varieties of warts and excrescences grow on the hands of pathologists and teachers of anatomy which are undoubtedly tubercular, but of a local character only, removable by surgical means. Therefore, a predisposition to the disease on the part of the individual is required as well as the reception of the germ virus.

*Method of Preparing a Cover-glass Slide of Bacillus
Tuberculosis.*

Take fuchsin, 1 part, and dissolve it in alcohol, 10 parts. Then add 100 parts of a 5 per cent. watery solution of carbolic acid.

Take a small portion of suspected sputa—the muco-purulent is best—and place it on a cover-slip, smearing the glass equably. Then pass the slip through the flame of a spirit lamp to fix the albumin. Place the cover-slip, or rather float it, face downwards, on a portion of the above fluid for five minutes. Drain off superfluous fluid, and place the slip in alcohol (90 per cent.) for a second or two, afterwards transferring it to a 25 per cent. solution of sulphuric acid, when the blue tinge of the fuchsin fluid is replaced by a yellowish brown. Then wash the slip in alcohol, and transfer it to a solution of lithium carbonate (1 in 20). Subsequently stain in a watery solution of methylene blue, clear with oil of cloves, and mount in Canada balsam. The bacilli will then be detected as red rods on a blue background.

A magnifying power of 400 diameters is required.

We may shorten the process by mixing the sulphuric acid with the methylene blue, the decolorising and contrast stainings being carried on at one time.¹

Pathology.—A tubercular deposit consists (say in the lungs) of small grey hard tumours about the size of a mustard seed. Each tumour is usually composed of groups of sub-miliary tubercles, and stands above the surface of a section of the organ. It is translucent when fresh, but soon becomes opaque, and is surrounded by an areola of inflammation. These tumours may remain discrete or may be confluent with their neighbours, and thus produce nodular masses with surrounding red areæ. These common masses ultimately caseate, forming yellowish growths which vary in size from a small pea to a walnut.

The microscopical structure of a tubercle consists essentially of small cells of an embryonic type. But the cells are

¹ See Woodhead's *Practical Pathology*.

arranged in a definite order, presenting three distinct zones. The outer zone is composed of leucocytes, which are the products of inflammation; the intermediary zone consists of epithelioid cells closely crowded together and consequently somewhat atrophied; whilst the central area consists of cells of larger growth, known as 'giant cells.' The giant cells are large irregular masses of protoplasm, with radiating processes, and contain numerous nuclei, chiefly arranged in a row at the circumference. They are usually situated near the centre of the tubercle, but not always. Consequently, in given microscopical sections they are not always apparent; the knife may have missed them, just as in certain slices of orange the pips are not always seen, as the section may not have passed through that part of the fruit where they are situated.

Surrounding the cellular elements, and apparently secreted by them, is a fibrillar material holding them together. Blood-vessels are seen at the circumference of the growth only: they become obliterated as they penetrate towards the interior; and hence the nodule becomes opaque at the centre on account of molecular destruction.

The tubercle after reception of the bacillus would, therefore, seem to spring from connective tissue, but only in that condition in which there is embryonic proliferation of its cells, so that a tubercular deposit is surrounded by such proliferation. For example, if it occurs in the interlobular tissue of the liver there is always a precedent interstitial hepatitis; if in bone, there is a preceding osteitis, and the formation of embryonic tissue in the medullary cavity; if in lung, there is first a proliferation of the interalveolar or peribronchial connective tissue, or a similar proliferation of the cells lining the alveoli.

Secondary Changes.—A tubercular growth subsequently undergoes one of three degenerative changes.

1. It may undergo caseous degeneration. This is not due to the obliteration of its vessels, as was once supposed, since syphilitic gummata caseate, although their blood-vessels remain patent. It is caused by the abundance and vigour of the bacilli, and the extent of the caseation is in direct ratio

thereto. This is the most typical change of tubercle, and constitutes the crude yellow tubercle of former pathologists. The change is a necrotic one ; the cells undergo fatty degeneration, their nuclei disappear, and the growth becomes opaque and structureless. It begins, and is best marked in, the centre, from which it extends centrifugally to the circumference.

A caseous tubercular mass usually liquefies and breaks down ; this change occurring not only in the component tubercles themselves, but in the whole growth and its surrounding area. Hence cavities are formed, the walls of which are lined with adjacent tubercles, which also act as advancing causes of fresh lesions.

Occasionally, however, as in the brain, a tubercular growth may remain caseous and not go on to liquefaction.

2. If the lesion is deeply situated in any organ, it may dry up and calcify, leaving an irregular or stellar cicatrix.

3. In a certain proportion of cases in which the bacilli are few and inactive, there ensues a fibrous degenerative change, the result merely of a chronic inflammatory process.

Tuberculosis may be **local** only, consisting of one focus, which may remain quiescent for some years and then gradually extend, or it may ultimately undergo calcareous or other degenerative changes of a beneficial character.

On the other hand, **extensions** of tubercular deposits may occur by means of the lymphatics (tubercular glands), by serous surfaces (pleura, peritoneum, &c.), or by ducts and tubes (trachea to bronchi, intestine from swallowing sputa, testes to vesiculæ seminales), or by means of the blood itself (general tuberculosis). In this latter state there are many centres of the disease, the condition being attended by general acute fever and rapid death.

Symptoms.—The two cardinal symptoms which accompany tubercular infection are fever and emaciation. The fever is usually of a hectic type, with profuse perspirations alternating with hot dry skin. It is due to the circulation in the blood of the bacilli and their putrefactive or decomposing products. Emaciation is secondary to, and caused by, the fever. All the

other symptoms depend on the locality of infection. Meningitis will occur when the brain is involved ; cough and expectoration in pulmonary deposits, albuminuria in renal tubercle. A general tuberculosis, however, closely resembles enteric fever in many of its clinical aspects, and not infrequently the one disease is mistaken for the other, especially in their earlier stages. Indeed, the subject of Tuberculosis should find a more suitable place amongst the Specific Fevers.

For a fuller description of symptoms refer to diseases of the various organs.

GENERAL DISEASES

GOUT

Definition.—A constitutional disease, characterised by increase of uric acid in the blood, and usually by the deposit of urate of soda in the joints, and the advent of chronic interstitial nephritis.

Causation.—(a) **PREDISPOSING.** *Age.*—A disease of middle and advanced life, occurring after forty in males, and after fifty in females.

Sex.—More frequent in men, on account of wear and tear of work and worry.

Climate and Season.—Most frequent in England, and during spring, with its cold, sunless skies and its east winds.

Heredity.—Strongly marked ; there appears to be a ‘basic, diathetic, or constitutional habit of body,’ from which springs gout on the one hand and rheumatism on the other the particular line of divergence being determined by other conditions, such as disorders of stomach, kidneys, &c.

Occupation and Habits.—It is said to prevail amongst lead workers, but statistics do not show this clearly. It is certainly prevalent amongst brain workers, and those whose mental powers are of a high order. The actual nervous system may, however, be feeble or irritable. It is therefore by some regarded as a neurosis—a functional disorder of a possible joint centre, situate in the medulla, or other part of the nervous system, but not necessarily a gross lesion. A sedentary occupation, involving much mental preoccupation or strain, certainly claims many victims. Amongst the

labouring class it appears to affect those who consume stout and porter; at least, this appears to be the firm conviction in the minds of Irish, Scotch, and Continental physicians. The disease is comparatively unknown on the Continent, whilst it is frequent in London, and, to some extent, in Dublin, where stout is a favourite beverage.

Nervous Derangement.—It is predisposed to by outbursts of rage, or by grief, worry, and anxiety.

Stomach Disorders.—Such as dyspepsia, a large meal, especially if accompanied by effervescing or sweet wines and liquors. High living in general is a potent predisposing factor, by overloading the digestive and excretory organs. There is usually in all gouty patients a feeble digestion. Oxaluria, as a result of dyspepsia, is a frequent antecedent condition.

Muscular Exercise and Injury.—Attacks frequently date from severe exercise, such as hunting, mountaineering, or from a blow or injury, especially in the immediate neighbourhood of a joint.

It will be seen in the foregoing list that many of the predisposing causes are linked together, such as sedentary occupation and indigestion. Probably one or more factors are required as producers. Again, some of them may be regarded almost as exciting causes; but the presence of an excess of uric acid in the blood is an essential.

(b) EXCITING CAUSE.—Uric acid is the irritant, usually in the form of urate of soda. It is over-produced by deranged function of the liver, and deposited locally in inflamed joints. Uric acid may also be formed in excess after exertion, or its excretion may be retarded, as in renal disease. W. M. Ord, on the other hand, regards it as a production due to the degeneration of fibroid tissues. As it circulates it gives rise to 'misemployment of nerve force, determining local paroxysms in one or more parts of the frame.'

'This may not necessarily produce gout, but will do so in a patient whose nervous system is ready to receive the impress of the irritation.' It will thus be seen that there is a neurotic factor and a humoral factor in its causation. Other

acids are present in the blood, such as lactic (Todd) and oxalic, both of which favour precipitation of uric acid, free or combined, whether it be in excess or not.

Pathology.—No other disease appears to have such varied and extensive pathological conditions. The pathology of gout is practically demonstrated in its symptoms; and as no organ or tissue appears exempt from disorder or change caused by gout, its morbid anatomy is really written in the descriptions of many other diseases. The lesions of joints are, however, the principal ones. Any joint in the body may be the seat of attack, but especially those of the lower extremities; then come, in order of frequency, the articulations of the hands, wrists, and elbows. On examining an affected joint we find a certain amount of synovitis, and, in addition, the articular surfaces are coated with white matter (urate of soda), not unlike the appearance produced by unskilfully opening an oyster, when the mollusc is smeared with pulverised carbonate of lime. The articular surfaces of both bones which constitute the joint are usually painted with the deposit, but an earlier and a thicker incrustation occurs on the concave surfaces than on the opposing convex surfaces of a joint. For example, urate of soda is invariably found on the concave surface of a first phalanx of the great toe, whilst it frequently is absent from the convex aspect of the terminal end of the first metatarsal bone. This, of course, only refers to mild cases. In the severer forms the whole joint may be disorganised by uratic deposit. The urates appear on the surface of the cartilages; if found in the deeper layers they probably extended from the bones. They are in the form of fine curved needles, and invade the matrix, being deposited there irrespective of cartilage cells and their capsules. As a result of this invasion, the superficial layer or zone of cartilage cells disappears, and is replaced by the incrustation. Consequent on the irritation, many of the conditions described in chronic osteo-arthritis are set up. The synovial fringes and membranes are thickened and impregnated with urates. The edges of the articular cartilages share in the inflammatory process, and develop into thickened enlargements; ‘lippings’ occur at the

circumferences of the bones, which, when examined microscopically, are found to be true exostoses; bony ankylosis takes place occasionally; finally, deflections of digits to the ulnar side, and other dislocations may occur. From the deeper structures the inflammatory process extends to the tendons, sheaths, and skin, in which also deposits of urate of soda gradually accumulate, until the joints present a nodular or spherical appearance, the skin over which is tense and atrophied, easily revealing the 'chalky' deposit of urates. Ultimately, unhealthy suppuration of the skin may ensue, and a slough or sinus may form, through which the 'mortary' compound may escape or be extracted.

There is a difficulty in assigning reasons why an attack of gout should be accentuated in one particular metatarso-phalangeal joint. Possibly it may be due to the effects of pressure and strain on the ball of the great toe, and this would coincide with the accepted doctrine that those parts which are injured or over-strained are just the ones fitted, by their impaired nutrition, to receive the gouty poison; in other words, gouty deposit only occurs in those tissues already degenerating—gout is a mode of decay of the system. For similar reasons we find it attacking the hands of mechanics and other working men, and especially the first left metacarpo-phalangeal joint, which is liable to injuries and blows from hammers and other tools.

The kidneys also show grave pathological changes, even if no other organ be affected. We have never opened a gouty joint, *post mortem*, without finding, on examining the kidneys, that they were granular in a more or less pronounced form. This observation is contrary to Sir Dyce Duckworth's experience, as, according to him, gout may occur without implicating the kidneys. The change may only consist of slight but unmistakable increase of the fibro-nucleated tissue between the tubules and vessels, or it may amount to the complete disorganisation of advanced renal cirrhosis. Interstitial and intertubular crystalline deposits occur, but are rare. Together with the renal changes we shall find sclerosis and thickenings of arteries, hypertrophy of the left heart, and all the cardio-

vascular changes found accompanying contracted granular kidneys. Other pathological changes occurring in different organs and tissues, although proper to many gouty subjects, do not invariably occur, and may therefore be referred to the lists of complications.

Symptoms.—By some authors gout is divided into two varieties at least, acute and chronic ; as though the symptoms were widely different in each. It would perhaps be better to regard gout entirely as a chronic disease, with occasional acute exacerbations or outbursts. It does not follow that, because a patient may have suffered from only one attack of acute gouty arthritis, he is cured, or that he is free from gouty symptoms in other parts. Prior to the acute exacerbation there are certain premonitory symptoms. The patient suffers from dyspepsia and acidity, with a copious deposit of lithates in the urine ; or he has headache with palpitation and increased arterial tension ; or he has headaches with irritability of temper and the like. Then, after retiring to bed, feeling somewhat out of sorts, he is suddenly awakened in the early morning by pain in the affected joint, most frequently the metatarso-phalangeal of a great toe. The joint is red, swollen, and exquisitely tender ; the skin over it is occasionally œdematous, and the neighbouring veins are swollen and turgid. The pain is so intense that he can tolerate no one coming near him, he cannot even bear the weight of the bed-clothes. His sufferings are not infrequently intensified by involuntary twitchings of muscles and startings of limb. There is more or less pyrexia (temp. 100° – 102°) ; the pulse is quickened (80 to 100) ; the bowels are constipated, the stools being pale ; the urine is diminished in quantity, and besides having the ordinary characters of a febrile state, it contains abundance of amorphous urates, and crystals of free uric acid. The nervous system is in a condition of great tension. The patient is irritable, restless, and quarrelsome, yet his mental faculties are clear and may be even sharpened. Grinding of the teeth is frequently observed during the height of a paroxysm.

After a few hours of fever, restlessness, and agony, his body becomes suffused with a rather free sweat, and he falls

into a slumber from which he awakes in comparative calm and comfort, but with the affected joint, or joints, red, swollen, and shiny. During the day he may be able to transact business from his couch, or even to hobble about with artificial aid. The next night, and probably the two succeeding ones, present repetitions of the previous attack ; they, however, diminish in severity, the febrile symptoms abate, and after a week or so, there is a return to usual health and condition of joint, the cuticle over which has meanwhile desquamated. It must be remembered that an attack is not necessarily limited to one joint, two or three may be involved at one and the same time. Indeed, though gout is regarded as most frequently involving the smaller articulations of feet and hands, the larger joints of the body may be affected, the hip alone appearing to escape. Further, during the acute outbreak, urate of soda can be detected in the blood.

Subsequent attacks as a rule occur—it may be in a week's time, or months or years ; the postponement depending on diet, exercise, occupation and other controlling influences. Each successive outburst is marked by an increase in the number of the joints affected, till ultimately the victim is in a condition known as chronic gout, with all its various complications of skin, stomach, lungs and other organs (*vide infra*). Occasionally, however, the explosion is sudden during apparent good health ; and it is difficult then to convince the patient that he is suffering from an attack of gout.

Authorities describe a form of gout, or a gouty condition in which there is a form of metastasis to internal organs ; as though the attack were not determined to the joints, or at least was arrested at a joint and then spent its fury on lungs, stomach, brain, or other part. This has been called 'Retrocedent' gout. The causation and the pathology of such a condition is not at all clear, but it is no uncommon thing for chronic gouty subjects to complain of symptoms referable to these organs, and to express their opinion that they would feel relieved if the gout would only come out into their joints.

Complications.—Some of the following may occur during

the acute stage, others are proper to the chronic condition ; or they appear as 'gouty manifestations' without any marked joint trouble ; for it must be understood that uratic deposits, even, may occur in an articulation without any of the usual local signs. Again, some may be regarded as symptomatic of the gouty state rather than actual complications. A patient once remarked on our explaining his state of health and complications, 'I am all gout !' This exclamation at least put the matter very tersely.

1. **CARDIO-VASCULAR.**—Besides the usual thickening of vessels and attendant hypertrophy of the left side of the heart which accompany contracted granular kidneys, there may supervene dilatation of the heart's cavities and calcareous degeneration of the aorta. Angina and anginoid attacks are not uncommon. Hæmorrhages may occur from nose, stomach, or other sites ; a gouty form of phlebitis is frequent, or thrombotic pluggings of large trunk vessels may be found.

2. **RESPIRATORY.**—Bronchitis is frequent, and is nearly always followed by emphysema. Occasionally pleurisy occurs, but a low, 'smouldering' form of pneumonia is more frequent, especially in debilitated subjects.

3. **DIGESTIVE.**—On examining the throat, the uvula is enlarged and pendulous, and is, together with the pillars of the fauces, congested and glazed as though painted with some oily substance.

Stomach disorders (pyrosis, flatulence, gastralgia) are so common as to be regarded as premonitory symptoms. They are usually produced by some errors of diet which precipitate the acute attack, and are vulgarly known as 'gout at the stomach.' Functional disorders of the bowels, such as obstinate constipation or irregular attacks of diarrhœa, may occur from time to time. Hæmorrhoids, ascites, and jaundice, secondary to cirrhosis of the liver, are apt to ensue. It is questionable, however, if the cirrhosis be so much a gouty manifestation as evidence of an alcoholic habit in a gouty subject.

4. **RENAL.**—Besides the signs of Bright's disease common to gout, we may meet with cystitis and urethritis, due to the irritating acid urine ; concretions (usually of uric acid) may

form in the kidney, bladder, or other part of the urinary tract. Glycosuria is so common as to be almost a constant symptom. With it there is polyuria, but not necessarily a high specific gravity of urine.

5. CUTANEOUS.—Many varieties of skin disease may complicate, or be significant of, gout. Tophi in the external ears, or in the eyelids, periosteum, or elsewhere may form; and not unfrequently they suppurate, producing unhealthy sores. The skin of the face is usually greasy, the capillaries, especially of the nose, are engorged; whilst troublesome forms of acne, psoriasis, or eczema, are common. The nails are usually coarse, and longitudinally grooved or fluted. Dupuytren's contraction of palmar fascia is often found in gouty mechanics, the pressure caused by tools and other instruments being probably the exciting cause.

6. NERVOUS.—Neuritis may supervene in any nerve trunk. Occasionally there are symptoms of meningeal inflammation, or the patient may suffer from hemicrania, or other forms of neuralgia. Insomnia is often distressing. Occasionally somnambulism occurs.

7. ORGANS OF SPECIAL SENSE.—*Eye.* A gouty form of iritis is universally recognised by ophthalmic surgeons. Retinitis with hæmorrhage, and glaucoma are occasionally seen, these probably being related to the renal changes. *Ear.*—Occlusion of the external auditory canal caused by œdema of skin, or exostoses of bone, or hypertrophy of sebaceous glands are the most usual forms of ear complication. Occasionally we find adhesion or ankylosis of the ossicles.

Diagnosis. 1. FROM RHEUMATOID ARTHRITIS.—In the acute stage of gout the diagnosis is easy. In confirmed gout we must be guided by a history of an acute outbreak; the presence of tophi in the ears or eyelids, of 'chalk stones' in the neighbourhood of joints, and of uric acid in the blood serum.

2. ACUTE ARTICULAR RHEUMATISM.—In this disease the inflammation selects the larger joints by preference; it is markedly metastatic; the fever is higher, there is copious sour perspiration, and finally a marked tendency to peri- and endocardial complications.

Appended is a table of the principal distinguishing points :

<i>Gout</i>	<i>Acute Articular Rheumatism</i>
Attacks one or two smaller joints	Selects larger joints
Remissions not common	Remissions common
Not metastatic	Markedly metastatic
Attacks middle aged	Attacks early adolescents
Attacks well-fed and robust	Attacks debilitated and poor people
Often connected with an error of diet	Often connected with cold and exposure

Treatment: I. DURING AN ACUTE ATTACK.—(a) *Local*. Support the affected limb on a pillow, or in a splint. Apply remedies to relieve the pain. Probably no disease has had such various and diverse local treatment. We should be guided by our experience, and also somewhat by the experience of the sufferer himself, if he has had a previous outbreak. Thus we may anoint the joint with extract of belladonna mixed with glycerine in equal parts; or apply the tincture of opium on lint. Or we may order hot fomentations with poppy heads, or paint the joint with a solution of hydrochlorate of cocain (gr. v. ad ℥ j). Some prefer the application of menthol dissolved in spirit; others depend on cold evaporating lotions (liq. plumbi subacet., with æther), the application of iced water. Others, again, prescribe poultices and fomentations. One or two leeches applied at some little distance from the inflammation may afford relief. We prefer to apply extract of opium on lint, and then to cover the joint lightly with cotton wool and a bandage. We must always be prepared with one or more remedies, as we are never sure which remedy will relieve; and we have to reckon with the caprice of the patient.

(b) *General*.—Give an emetic if the stomach be still loaded with an indigestible meal. A brisk purgative is always advisable, such as blue pill (gr. vj.), or calomel (gr. iv.), or compound senna mixture (℥jss) or sulphate of magnesia with sulphate of potash (each gr. xv.). When the bowels have acted freely colchicum is the best remedy. It is almost regarded as a specific. The vinum colchici in m℥ xx. doses every

four hours is the most effective.¹ In elderly subjects the preparations of salicin may be tried. The salicylate of soda, on the whole, is the most reliable. Sir A. Garrod recommends salts of lithia. The carbonate of lithia (gr. v. to x. three times a day) will at times give good results. The diet should be sparing, and consist mainly of milk, eggs, and farinaceous foods. Animal food, especially butcher's meat, should be avoided, or allowed in small quantities only. Alcohol is to be prohibited, unless symptoms of cardiac failure are present.

II. BETWEEN THE ATTACKS.—The treatment is mainly one of diet and regimen. Forbid sweet or highly-seasoned dishes. Big dinners and over-feeding in any way are injurious. In short, the dietary should be regulated in quantity and quality as though for a confirmed dyspeptic. Exercise in moderation is beneficial, but should stop short of fatigue and over-exertion. Mental fatigue and exhaustion are also to be avoided. Further, it is necessary to insist on a free action of the kidneys, bowels, and skin, by which means uric acid and its compounds may be eliminated. We may therefore advise Turkish and hot-water baths; we may encourage the imbibing of large quantities of warm water or other diluents. An occasional course of iodide of potassium in combination with bicarbonate of potash is valuable. Sometimes insomnia is a distressing condition. It usually yields to a full dose of calomel (gr. iv.). Finally, if the patient's means and time allow, we may send him to Bath, Buxton, Carlsbad, or other health resort, or put him under care at some hydropathic establishment where he will usually derive benefit, not so much from the chemical nature of the waters, as from freedom from care, the change of scene and surroundings, and the discipline which is enforced.

ACUTE ARTICULAR RHEUMATISM (RHEUMATIC FEVER)

Definition.—An acute, probably specific fever, characterised by inflammation of joints, tendons, and muscles,

¹ R. Vini Colchici m. xx.; Mag. Carb. gr. x.; Potas. Bicarb. gr. x.; Aquæ Menth. Pip. ʒj. Miscæ.—*St. Thomas's Hospital Pharmacopœia.*

profuse sour sweats, and a tendency to peri- and endocarditis.

The so-called sub-acute rheumatism is merely a less severe form of the acute.

Causation.—(a) PREDISPOSING CAUSES.

Age, most frequent between twenty and thirty ; but all ages liable.

Sex, has apparently no influence : some statistics give a preponderance of cases to males, others to females : probably the severe form is more frequent in men, and the sub-acute in women.

Climate and Seasons.—It prevails in cold damp climates, and in this country is most prevalent in October, November, and December.

Occupations.—Those involving exposure, or frequent changes from heat to cold, such as smiths, butchers, furnace-men, and the like. It is also found in persons who are ill-fed and overworked ; hence many cases occur in lodging-house servants, waiters, and the like.

Heredity.—Strongly marked : a child of rheumatic parents is more liable, in the proportion of 5 : 1, to acquire the disease than the offspring of healthy ones. This predisposition is most apparent when statistics are taken of young subjects : it is less marked when adults over fifty years of age are considered.

A Bad State of Health.—It is more prone to attack those of lowered vitality ; hence frequent in alcoholic subjects, in those who cannot obtain sufficient food, or rest ; and it often occurs in women from overlactation.

Specific Fevers.—Notably scarlet fever, ague, diphtheria.

Type and Configuration.—Our experience leads us to think that there is a peculiar type of males liable to acute rheumatism. They are of heavy, flabby build, with a tendency to obesity and consequent lethargy. They are apt to consume large quantities of fermented malt liquors. They not unfrequently are myopic.

Epidemics.—Acute rheumatism appears to prevail occa-

sionally in epidemics, which vary in character as to extent and severity of symptoms.

(b) EXCITING CAUSES.—1. The *virus* causing the disease has not yet been ascertained ; by some lactic acid is supposed to be the potent factor, but pathological evidence tends to negative this. 2. *Bodily fatigue and mental exhaustion*. 3. *Local injury*, especially to joints, or to the neighbourhood of joints. The disease appears first in that joint which is injured.

Incubative Period.—Not constant. It may vary from twenty-four hours to some days.

Pathology.—The joints which are involved show signs of synovial hyperæmia, a similar condition affecting the skin and the sheaths of tendons. The synovial fluid is increased in quantity, it may be turbid, and often contains flakes of lymph. The cartilage cells are proliferated, both in the superficial and in the deep layers. The inflammation never (or rarely) goes on to suppuration.

Pericarditis and endocarditis, though so common as to be almost symptomatic of the disease, are not necessarily present ; their pathology, therefore, is referred to these respective conditions.

The blood contains a great excess of fibrin, greater or less according to the height of the fever. In no disease is it in such excess except pneumonia. During the progress of the disease the quantity of blood corpuscles is diminished.

Symptoms.—The onset is gradual. It usually commences with a ‘feverish cold,’ accompanied by malaise, which is followed by flying pains in the limbs, back, and trunk, accompanied by a feeling of having been beaten with a heavy stick all over the body.

In about twenty-four hours the condition of the large joints shows the true nature of the disease. They are swollen, tender, and painful ; the synovial capsules are distended and tense, the sheaths of tendons are often equally painful ; the skin over the joints is œdematous and often red, but not always ; the pains are increased by warmth and by movement ; the muscles themselves are occasionally painful, and hence the

patient lies characteristically helpless on his back. The articular inflammation does not go on to suppuration.

The joints are not all affected simultaneously. Usually the larger ones are primarily and most severely involved. There is, however, no symmetrical invasion of joints; the disease is typically metastatic, being most severe (say) in one knee to-day, whilst to-morrow a shoulder or a wrist-joint or the pericardium may be most inflamed; and thus it flits, as it were, from place to place until the fever appears to be spent.

The skin is usually sodden and bathed in copious perspiration, which becomes acid after a time, but which is not distinctly acid when freshly excreted. It has a characteristic sour odour, noticeable to the attendant and patient. Sudamina are common. The temperature is high, ranging from 101° to 105° or higher: and it is distinctly remittent in type, the variations, however, depend on the severity of the attack, and the number of joints involved. The pulse (90° to 120°) is full, bounding, soft and regular, unless the heart be seriously embarrassed, when it becomes feeble and intermittent. The tongue is large, flabby, and coated with heavy creamy fur; the bowels are constipated. The urine is febrile: that is to say it is acid, high-coloured, and scanty, with its urea and uric acid increased. The fibrin in the blood is increased. In women the catamenia may be suppressed, but oftener antieipated.

The disease has no regular course; it may last from eight to fourteen or twenty-one days; sometimes it even extends to five weeks. There is, therefore, no critical day, the fever gradually ending in lysis.

Varieties.—It is found in some cases that the symptoms are not quite in accordance with the above description. For example, in (i) *sub-acute rheumatism* the fever is not so high, as the joint troubles are less pronounced, the sweats are not so copious, and there is not the helplessness and exhaustion of a typical acute attack. It is, nevertheless, not a distinct type, but merely a modification, and stands in the relation to rheumatic fever which scarlatina does to scarlet fever. The sub-acute variety, however, is liable to exacerbations, when all the

conditions of the acute attack may present themselves. (ii) *In children* acute rheumatism may present various modifications. Thus, it may appear as chorea, attended by feverishness, and often by joint pains, and followed by characteristic cardiac lesions. Or it may be characterised by the formation of subcutaneous fibrous nodules, attended by fever, and contemporary with the appearance of physical signs of endo- or of pericardial inflammation, and of arthritis. These nodules are undoubtedly manifestations of acute rheumatism in young subjects. They occur frequently in the offspring of rheumatic parents ; they may accompany all the typical symptoms of rheumatic fever ; or, with heart lesions, they may constitute the only manifestations of the disease. Their most frequent site is about the elbows, knuckles, knees, and superficial tendons, but no part of the body where fibrous tissue is well developed appears to be exempt. Further, it must be noted that in children the temperature and the joint affections are less, whilst the heart lesions are more accentuated than in adults. The question whether *Erythema nodosum* is a rheumatic manifestation or a complication only, is a debated one. Our experience inclines to the former view. (iii.) *At different seasons*, or during different epidemics, the cases may be marked by a preponderance of joint affection, with high fever, but with less pericardial or endocardial inflammation, whilst another series of cases will be characterised by symptoms just the opposite.

Complications.—These are tabulated according to the various systems involved. Some, notably heart affections, appear to be purely symptomatic ; but, as they are not always present or consequent, they are rightly regarded as complications.

1. CIRCULATORY SYSTEM.—*Heart disease*, whether involving pericardium, endocardium, or muscular walls, is the most frequent. At least fifty per cent. of cases are affected. Endocarditis is most frequent ; after that comes pericarditis. By some it is thought that pericardial inflammation is most common. This error may be accounted for by the signs of an inflamed pericardium being more palpable and superficial, and by the fact that endocarditis may exist and yet be masked by

the noisier physical signs of pericarditis. In addition, endocarditis may supervene suddenly (when convalescence has been apparently established) by the effects of strain or exertion on a valve which was damaged and had not recovered, even though it gave no auscultatory evidence of inflammation. In some, an endocardial affection may assume, apparently without cause, a malignant type (see Ulcerative Endocarditis).

2. RESPIRATORY SYSTEM.—*Pleurisy*, especially left-sided, may occur. In some instances it appears to be an extension from an inflamed pericardium ; in others as a direct result of the febrile process. The same applies to *Pneumonia*, but this complication is not common.

3. DIGESTIVE SYSTEM.—*Sore throat* (tonsillitis, pharyngitis) is frequent. Some regard the affection of the tonsils as quite a symptom of the disease ; and, remembering the association between pharyngeal and joint affections, there appears some reason for this contention (see Scarlet Fever, &c.). *Peritonitis*.—Occasionally, but rarely, inflammation may occur in the peritoneum, either as an extension from the pericardium through the diaphragm, or as a separate lesion.

4. NERVOUS SYSTEM.—*Hyperpyrexia* occurs in severe cases, the temperature rising as high as 105° or 107°. Such a high temperature alone would constitute a grave condition, whatever its cause. *Delirium*.—Most frequently in males, and in severe pericarditis. When accompanying pericarditis, it has in some cases been more pronounced and 'wild' than in any other disease we have seen. *Chorea*.—Frequently in children, but rather as a sequel than a complication. It may, however, supervene during the attack. *Peripheral neuritis*, especially of the extremities, manifested by pain and tenderness of hands and feet, atrophy of muscles (interossei, thenar, and hypothenar eminences, etc.). The joints which are moved by the affected muscles are not necessarily involved.

5. EYE.—*Glaucoma and Iritis*.—Rheumatic iritis is said to be one of the commonest forms.

6. CUTANEOUS SYSTEM.—*Erythema nodosum*, especially in debilitated young subjects, or *Urticaria*, may appear during an attack ; or, if there is no other personal evidence, they

frequently occur in patients who have a marked family predisposition to rheumatism.

7. **RELAPSE.**—A return of the fever with all its signs and symptoms, including fever, perspiration, and joint pain. Endo- and pericardial affections, which may not have existed in the initial attack, may complicate a relapse.

Other rare complications are nephritis, jaundice, orchitis, sclerotitis, and inflammation of the meninges.

Prognosis.—The mortality from acute rheumatism, apart from its heart complications, is low, not exceeding three to five per cent. It is pericarditis which kills during the attack, but ultimately endocarditis and its effects are the most fatal. Or, during a relapse or second attack, an endocardial affection, running an apparently normal course, may suddenly become malignant (pyæmic) and destroy the patient. Pneumonia and delirium would also seriously affect our prognosis.

Diagnosis.—1. **FROM PYÆMIA.**—In this disease the odour of breath and of perspiration is sweet and pungent ; endo- and pericardial inflammations are rare ; there is no metastasis—often one joint only is involved. Inquire for previous suppuration, periosteal trouble, or surgical operation.

2. **FROM GOUT.**—This disease mostly affects adults past middle age. The smaller joints are usually involved, especially the metatarso-phalangeal articulations of the great toes. The presence of tophi in the ears, of uric acid in the urine, and the family history of gout, would be conclusive.

3. **IN GONORRHOÆAL RHEUMATISM** the joints involved are fewer, the disease is obstinate, and yields less readily to treatment. There is a history of urethral discharge.

4. **IN CHRONIC SYPHILIS** there are periosteal nodes, and pains, which are worse at night. The symptoms yield to biniodide of mercury.

5. **IN DYSENTERY, SCARLET FEVER, and LESIONS OF THE CENTRAL NERVOUS SYSTEM,** the inflammations are not so acutely centred in the joints. There would, in addition, be a history of dysentery, or of sore throat, followed by desquamation, or of injury or disease of the spinal cord, trunk, nerves, &c.

Treatment.—GENERAL.—Absolute rest in bed is imperative. This applies especially to sub-acute rheumatism ; in the acute form the patient is, of course, too helpless to stir. The bed should be firm, covered with a good layer of blankets ; the patient should lie between blankets, and dressed in a flannel garment ; linen next the skin is injurious, as it fails to absorb the excessive sweats, and hence is cold and uncomfortable. He should have a nurse who is able to lift him, and so be secured from any exertion whatever. His body may be occasionally sponged with tepid water, with marked relief.

The affected joints should be protected by flannel rollers, or by splints, or by layers of cotton wool, on which opiate preparations may be sprinkled if necessary. Occasionally, great relief is afforded by freely blistering the skin over or in the neighbourhood of the joints. It can do no harm, and patients frequently affirm that it relieves the pain and tension. The diet should consist of milk, eggs, meat-broths, and soups. The patient may be encouraged to drink freely of water, and bland, non-alcoholic beverages. The bowels should be regularly relieved, a preliminary purgative, such as calomel (gr. iij.) or castor oil, being given.

MEDICINAL.—For the rheumatic affection itself, the introduction of salicin and the salicylic compounds has been a great advance in medical therapeutics. Dr. MacLagan appears to prefer salicin, and holds that the salicylic drugs are distinctly anti-rheumatic, and therefore specific remedies, with which our experience leads us to entirely agree. The exact preparation which is most useful does not appear to be definitely settled, some preferring one form, others advocating a different one. Probably the efficacy of the different preparations depends on the idiosyncrasy of the patient to a great extent. We prefer the salicylate of soda (gr. x.) every three hours. It is wonderfully assisted in its action by bromide of ammonium (gr. x.), which helps to relieve pain and secure sleep.¹ If the salicylates fail, we should try salicin (gr. v. to xx.), or salicylic acid (gr. v.). The latter, however, is rather depressing.

¹ R. Sodæ Salicylatis gr. x. ; Ammonii Bromidi gr. x. ; Syrupi Aurantii mxx. ; Aquæ Camphoræ ʒj. Misc.

The effects of these drugs are to lower temperature, to relieve the joint pains, and to cut short the course of the disease. Some physicians prefer one or two large doses at the commencement, followed by smaller doses, which are continued for a week or more after the temperature has fallen to normal. The results of all these preparations are similar to that of quinine, and they should be discontinued if delirium supervene, unless it be due to hyperpyrexia.

Yet, some physicians still adhere to the old alkaline treatment, and their statistics do not appear unfavourable. They give nitrate of potash, or the bicarbonate, or the acetate either separately or in combination. A good plan is to give the patient a solution of nitrate and of bicarbonate of potash (each gr. v. to ʒj) and allow him to drink it freely. Cases thus treated are said to have less frequent cardiac complications. The pulse rate is certainly lowered, which, with threatened endocarditis, is an advantage. Others prefer the exhibition of quinine in full doses with bicarbonate of potash. Or they give tinct. aconite (m. iij every four hours); it causes temporary anæsthesia and subsidence of febrile symptoms, but does not appear to permanently relieve the rheumatism (Peacock). But most authorities agree that the salicylate compounds should be tried first.

Antipyrin (gr. v. to xv.) is also useful, especially when the temperature threatens to rise above 103°. It has the advantage of not causing the distressing deafness and other ear symptoms of quininism.

In hyperpyrexia the application of cold to the surface produces marvellous results. In private cases we may apply ice or iced water packs; or, in hospital practice, where nursing help and appliances are at hand, we may give the graduated bath (90° lowered to 65° in fifteen minutes). Not only is the fever diminished, but delirium is controlled, and even removed. It is said that lung complications do not necessarily contraindicate the application of cold to the surface. We should doubt this. Certainly, the existence of lung disease in enteric fever would make us hesitate to order the graduated bath.

Opium and its derivatives may be given to ensure sleep. Dover's powder (gr. x.) is, on the whole, the best preparation.

Rheumatic pericarditis requires no special treatment apart from the ordinary rules (see Pericarditis). In strong plethoric subjects, however, it is beneficial to apply leeches (six) over the pericardium, or to apply a large blister. The objection to the latter procedure is that it prevents daily stethoscopic examination. This difficulty, however, is overcome by using the binaural stethoscope. If pericardial effusion be so great as to embarrass the heart's action, and so threaten life, we should not hesitate to perform paracentesis. A stout hypodermic needle should be inserted at the left side of the sternum in the fourth space. The needle should be lateralised so soon as it enters the pericardium, so as not to injure the heart. Aspiration then will easily remove sufficient of the fluid.

For endocarditis rest is essential. And it is imperative that the period of repose should be continued for weeks or months if possible, after all signs of rheumatism have disappeared. It will frequently happen that a patient dismissed as cured, and with no sign whatever of valvular disease, will return some months later with a marked mitral or other valve lesion. We think that these lesions might have been obviated by prolonged rest. The rest which is enjoined during convalescence is of more importance from the cardiac aspect than from any fear of return of arthritis. Subsequent anæmia demands the administration of iron in large doses.¹

CHRONIC ARTICULAR RHEUMATISM

Definition.—A disease due to the rheumatic diathesis characterised by chronic inflammations and destructive lesions of the joints.

Causation.—1. *Age.*—Most frequent after middle life. 2. *Sex.*—Both suffer; but men more frequently than women, owing to their exposure to cold and wet. 3. A previous attack of *Acute Rheumatism*; in many cases, however, there is no such

¹ See Dr. A. W. Garrod, *Treatise on Rheumatism*.

history. 4. *Climate and Season*.—It prevails in cold, damp, moist countries, during the autumn and winter. A sudden change from warm weather to cold will induce an attack. The patients regard themselves on this point as trustworthy barometers. 5. *Occupation and Station*.—It occurs amongst those who are exposed to vicissitudes of climate. Hence seen in cabmen, river-side labourers, sea-pilots, washerwomen, &c. It is also favoured by poverty, scanty clothing and nourishment.

Pathology.—The cartilages, synovial membranes, and fibrous capsules of the affected joints are thickened and roughened either by previous attacks of acute rheumatism or by the frequent recrudescence of chronic arthritis. The pathological changes may be limited to the above. But, on the other hand, they may be so extensive as to present almost complete destruction and absorption of the cartilages, tendons, synovial fringes of the joint, and even the formation of hard, bony layers on the opposing surfaces, together with rings or ‘lippings’ of bony tissue at the circumferences, as in other forms of chronic arthritis. Chronic valvular disease may exist, or it may be entirely absent. If there be any valvular lesions they are indistinguishable from the various forms due to chronic endocarditis. ‘The attack is always attended by a considerable diminution of the number of red corpuscles.’

Symptoms.—Vary in intensity and character in different subjects. There appear to be at least two varieties. In those cases in which there is *no history of previous acute rheumatism* there is pain, stiffness, and at times partial dislocation of joints. The patient has difficulty in moving; each movement of the affected joints is attended by increased pain, and by a peculiar grating or creaking sound which can be heard easily on auscultating directly over the articulation.

The joints most frequently involved are the larger ones, such as knees, shoulders, wrists; but this will depend in no small degree upon the occupation of the patient. It would appear that those joints are first and most severely affected which are subject to exposure, or to injury, or to strain. Thus, it is most marked in the hands and wrists of laundresses,

in the shoulders of cab and omnibus drivers, and in the knees and ankles of domestic servants.

These symptoms occur from time to time, with intervals of comparatively good health and freedom from pain. The return of symptoms is usually associated with some marked inclement change of the weather. These may be the only symptoms. The disease is a local chronic arthritis, and nothing more, occurring in those prone to take on rheumatism.

In another class of cases *having a previous history of acute rheumatism*, the conditions may be regarded as sequelæ with exacerbations rather than manifestations of a fresh disorder. Similarly, the diseased joints are stiff and creaking; they often present a distinct crackle or crepitus on passive movement: the capsule and the skin over it are occasionally red and swollen; recrudescences of arthritis, till they almost amount to the subacute variety, are not uncommon, and the muscles in the immediate vicinity are feeble and wasted. The disease may cause dislocation of joints, or even the marked structural changes of rheumatoid arthritis.

Occasionally, but not always, there is valvular disease of the heart, the result of the previous acute attack. Some observers, however, appear to think that a chronic endocarditis, with thickenings and distortions of valves may exist *pari passu* with the chronic arthritic trouble. It would seem more probable that in chronic rheumatism the disease, instead of spending itself on the heart and joints (as in acute), is of a less severe type, and exhausts itself in attacking the joints only, from time to time.

In addition, the patients are usually feeble; nearly always anæmic; their digestive powers are impaired, and their mental condition is irritable or morose.

Treatment.—In the majority of cases, there is no complete cure. We can, however, alleviate the various conditions considerably. The main indications are:

1. RELIEVE THE PAIN.—The patient should wear flannel next the skin, and rollers of flannel round the affected joints. New flannel has a good reputation, possibly on account of the sulphur which it contains. We may order opium, belladonna,

and other anodyne applications to the joints. Friction and passive movements afford relief. Stimulating liniments of turpentine, or chloroform are useful, patients having immense and justifiable faith in an embrocation applied directly to the diseased part. Salicylates are of no use except in chronic exacerbations dating from an acute attack. Iodine liniments, however, are often of immense service.

2. POSTPONE AND DELAY THE ATTACKS.—This is best done by improving the general health. The diet should be generous and nutritious. Alcoholic stimulants, with the exception of fermented liquors, are not necessarily to be forbidden. Iron, cod-liver oil, are indicated in weakly anæmic subjects. Arsenic is especially valuable in old people. In a series of cases we find no remedy of special advantage over the others; some being benefited by one drug, others by a totally different one. We may, therefore, try the effects of iodides, and bromides, guaiacum, sulphur, and the various preparations of iron. The patient should be warmly clad, but removal to a warm climate does not appear to be always beneficial. In addition, frictions, massage, and passive movements of the affected joints relieve pain and restore movement. We suspect that the reputation which many embrocations enjoy is in no small degree due to the rubbings and movements of joints which they entail.

The various baths and watering places which are in repute are: Buxton (warm), Bath (hot and ferruginous), Droitwich (brine), Strathpeffer (sulphurous), Aix les Bains (warm and sulphurous).

More than half the cure, however, of these health resorts is due to the change of air, scenery, and surroundings, together with the regulated healthy dietary, regimen, and discipline of the institutions which are visited. If possible select a residence on a gravel soil.

OSTEO-ARTHRITIS

Definition.—A comprehensive term applied to many different conditions and diseases, all of which are mainly characterised by inflammation of the joints, including bones, cartilages, synovial membranes, and fibrous structures.

Causation.—A classification of causes is difficult, and at best arbitrary. In the following table the principal conditions only are enumerated.

1. RHEUMATIC : (a) *Acute* ; (b) *Chronic*.

2. GOUTY.

3. INJURY : (a) *Direct* ; (b) *Indirect*, from traumatism of some neighbouring part.

4. DIRECT SPINAL IRRITATION : (a) *Of cord or medulla* ; (b) *Of dura-mater (pachymeningitis)* ; (c) *Of nerve roots*.

5. REFLEX SPINAL IRRITATION : (a) *Uterine irritation* ; (b) *Prostatic irritation* ; (c) *Urethral irritation (Gonorrhæal)* ; (d) *Vesical irritation (Calculus, &c.)*.

6. RHEUMATOID : ('*Arthritis Deformans*').

In (1) rheumatic and (2) gouty arthritis the lesions are local manifestations of a general disease, whilst the conditions named under headings 4, 5, and 6, might perhaps be grouped together under nervous disorders, although their causation widely differs.¹

Pathology.—In all chronic forms there are erosions of cartilages, a tendency to the formation of osteophytic outgrowths at the circumferences, thickening of synovial fringes, and eburnation of bone. In the spinal column these outgrowths are often so pronounced as to overlap neighbouring vertebrae, fixing them together, not so much by true ankylosis as by invasion or overflow of bony tissue, resembling the gutterings of a wax candle ; these are especially well seen in the bodies of the vertebrae.

These may be the principal or even the sole local manifestations of disease. This evidence, together with certain symptoms have led some physicians to regard arthritis in many

¹ See W. M. Ord, Address to Brit. Med. Assoc. Belfast, 1884.

of its varieties, not so much as a systemic disease, as a result of disturbed nutrition, a dystrophy of joints, due to local or central nervous irritation. Joint lesions similar in character are found in diseases which are quite different in symptomatology, such as locomotor ataxy and rheumatoid arthritis.

Other joint changes, such as occur in gout and acute rheumatism, are described under their respective headings.

GONORRHEAL ARTHRITIS

Definition.—A form of synovitis which occurs as a sequel to gonorrhœa.

Pathology.—It has been regarded by some authorities as a disease akin to pyæmia; but the slight constitutional disturbance and the absence of suppuration tend to negative this view. It would seem to be more probably a reflected dystrophy of joints, secondary to prostatic or to uterine irritation, conveyed to a centre in the spinal cord.

Symptoms.—There is a previous history of gonorrhœa, or of uterine or vaginal discharge. The disease appears to specially affect thin, weakly subjects. The symptoms are mainly those of sub-acute rheumatism, limited to one or two of the large joints, the knees or elbows. The changes are for the most part limited to the fibrous tissues of the joints, which become swollen, red, and tender. But pains in the plantar fasciæ, and in the sclerotics, are not uncommon. The inflammation subsides in a few weeks and does not recur, as a rule; unless there be a newly acquired urethral trouble. The joints remain stiff for many weeks or even months after all active mischief has ceased.

Diagnosis.—1. From SUB-ACUTE RHEUMATISM, only by the evidence of gonorrhœal discharge.

2. From GOUT, by the youth of the patient, and the absence of gouty evidences in the urine and cardio-vascular system.

Treatment.—Arrest the urethral or the vaginal discharge. Perfect rest is an important factor in the cure. The joints should be freely painted with iodine and placed in plaster of

Paris or other form of splints. Good nourishing food is also necessary. Advantage will also accrue from the administration of cod-liver oil with iron, quinine, and other tonics.

RHEUMATOID ARTHRITIS (ARTHRITIS DEFORMANS)

Definition.—A form of chronic arthritis, occurring in advanced life, characterised by distortion and dislocation of the joints, especially of hands and feet, and apparently associated with, if not consequent on, disturbances in the central nervous system.

Pathology.—The lesions are confined to the joints and to the muscles, and the skin in their immediate relation. The cartilages have a velvety appearance due to the matrix breaking up into fibrillæ: they become worn and even absorbed at the points of greatest contact and pressure. The bony elements become hard, polished, and ivory-like, and eventually exostoses form round the circumferences of the articular surfaces, so much so that the character of the articulation is entirely altered, a convex globular head being transformed by the osteophytic outgrowth as well as by attrition, into a concavity. These changes are most pronounced in the smaller peripheral joints. The synovial fringes are thickened into tuberculated clusters which may become cartilaginous and get detached. The periosteum, ligaments, and tendons in the vicinity of a joint are thickened and occasionally absorbed. There is usually an increased amount of synovial fluid. The muscles which move the joint are weakened and atrophied.

The remote pathology is not as yet made clear. Charcot regards it as a form of chronic rheumatism. Another good observer (Hutchinson) looks upon it as a mixture of gout and rheumatism, in persons who have an inherited tendency to arthritic diathesis; and ‘so long as an arthritic person has sound digestion and healthy kidneys, so long will his rheumatic manifestations be free from gout.’ In other words, the two conditions may mingle as easily as alcohol and water.

Again, Ord, Duckworth, and A. E. Garrod, assume, on clinical grounds mainly, that it is a pure neurosis, a defect of nutrition of the joint, and that the changes observed rank, therefore, as trophic ones. When we consider the various groups of 'causation' and the clinical evidence, this latter view appears to us the more scientific and correct.

Causation.—1. AGE.—Most frequent between forty and sixty years, dating from the menopause in women, and from the period of early degenerative tissue change in men.

2. SEX.—By far the greater majority of sufferers are women : probably in the ratio of three to one.

3. HEREDITY is marked to a slight extent ; but in most cases there is a strong arthritic family history, whether it be the gouty, rheumatic, or other form. This would tend to point, therefore, to its neurotic origin.

4. UTERINE DISORDERS of all characters, such as menorrhagia, dysmenorrhœa, uterine fibroids, the varying forms of endometritis, &c. *Menopause* also has an important bearing on its causation, probably as a reflex irritation. A similar remark applies to *rapid childbearing*, especially in cases of privation and overwork. Another most important predisposing factor is *prolonged lactation*. It is possible also, that as it appears in those who have been subjected to prolonged care and nursing of the sick, and in women who over-suckle their offspring, cases in this category should be referred to mental strain (5), and lowered vitality (6), respectively.

5. MENTAL STRAIN, due to shock, grief, or anxiety.

6. LOWERED VITALITY.—Hence frequently seen in the out-patient rooms, amongst people who suffer from poverty and privations.

7. INJURY, either direct to joints involved, or from lesion or irritation of spinal cord, including its membranes and nerve roots.¹

Symptoms.—The onset is gradual and insidious. The affection mostly begins in the joints of hands and feet, especially the former. It then extends upwards to the larger joints,

¹ See W. M. Ord : *Neurotic Dystrophies*, Brit. Medical Assoc., 1884.

wrists, elbows, ankles, and knees. Before the terminal small joints are structurally involved, there is a complaint of numbness and tingling, followed by pains in the joints themselves, in the bones constituting the joints, and in the muscular eminences of the hands and feet. There is marked symmetry of invasion on the two sides, whether the hands or the feet are primarily affected ; and also there is a decided symmetry in the appearance and amount of the arthritis. The temporomaxillary articulation is also involved early, and, according to Sir Dyce Duckworth, this is almost diagnostic. The pains in the joints are described as 'gnawing' in character ; they are increased by movement, and aggravated by warmth in bed.

In addition, the pulse rate is quickened, the tension increased ; and there is a tendency to freckles, and other pigmentary changes in the skin (Kent Spender).

As the disease progresses, the joints are enlarged, the knuckles becoming swollen and nodular ; there is synovial effusion ; the shafts of the bones—phalanges especially—are wasted, so that each finger represents a series of spindles and nodes, the internodular spaces being represented by the attenuated shafts. Subsequently there is a tendency to deflection of fingers, the terminal phalanges deviating to the radial, whilst the first and second rows are dislocated towards the ulnar side. Concurrently with these signs the muscles of the affected joints are wasted ; they are subjected to painful spasmodic contractions ; the skin over the fingers is thin and glossy (atrophy) ; the various tendon reflexes are increased.

In extremely chronic cases, similar changes occur in the joints of the spinal column, and in the larger ones of the limbs (shoulders, hips, &c.), this condition being attended by rigidities. The trunk is flexed, the thighs drawn up on the abdomen, and the upper limbs are flexed, adducted, and pronated. Various types of further deformities of hands supervene, and appear to depend on the wasting of certain muscles and the integrity of others. The second rows of the phalanges are, however, generally extended, and this may be accompanied by flexion or by extension of the other rows.

There are no evidences—or very rarely—of cardiac lesions of acute rheumatism, unless this disease occurred as a previous illness, or as a complication.

All the symptoms are liable to periods of quiescence, with alternations of exacerbation which are mainly influenced by weather.

Prognosis.—Good, so far as life is concerned. Many patients live years, but years of pain and helplessness. Death is usually caused by exhaustion, or by some intercurrent disease, due to want of exercise, or to deficient circulation.

Diagnosis.—It may be confounded with the joint lesion of locomotor ataxy.

Charcot's Disease

Rheumatoid Arthritis

1. Pain slight.	1. Pain severe.
2. Much effusion.	2. Little or no effusion.
3. Dislocations and fractures common.	3. Dislocations and fractures uncommon.
4. Bones atrophied.	4. Bony outgrowths.
5. Affects knee and shoulder.	5. Affects hips and smaller joints.

Treatment.—1. GENERAL.—Rest and a generous diet are the first essentials. It is noteworthy how rapid is the improvement in patients admitted to hospital, where they are kept in bed, and their affected joints bandaged or otherwise restrained from movement. A full dietary of animal and vegetable foods should be given. Alcoholic stimulants in moderation are not contra-indicated. Marked benefit often accrues to exhausted patients by their having ale, or even port wine, at their principal meal. Arsenic frequently works wonders in old anæmic patients. Cod-liver oil and tonics are indicated when the patient is emaciated and feeble. Iodide of potassium should be tried, especially when the pains are aggravated at night, or with warmth.

For insomnia, due to pain, give the bromide of ammonium, which may be combined with iodide of potassium (gr. iij.) and salicylate of soda with advantage.

2. LOCAL.—Stimulating liniments appear to give general relief. The patients themselves ask for an embrocation, so it

is only reasonable to infer that they find relief by their use. We have experienced good results from the use of belladonna, either in the form of the liniment, or the extract mixed with an equal part of glycerine. Recently the oil of gaulteria has been advocated. When mixed with an equal part of olive oil it forms an excellent liniment, but the results vary ; in some it affords almost instant relief.

The question of health resort is a difficult one, and will be influenced by the means and time at the patient's disposal. Continental watering-places are, on the whole, most advisable, such as Carlsbad, Homburg, in cases complicated with dyspepsia, and Spa, in anæmic cases. Other beneficial resorts are Bath, Buxton, and Strathpeffer, and the mild climate of Algiers and Morocco.¹

MUSCULAR RHEUMATISM (LUMBAGO, PLEURODYNIA, WRY NECK)

Definition.—A disorder occurring mostly in rheumatic subjects and characterised by severe pains, with contractions in the muscles, or in a group of muscles, accompanied by slight fever.

Lumbago

Causation.—There is usually a predisposing condition, such as the rheumatic or the gouty diathesis. It occurs also frequently in patients who are accustomed to an excess of animal food, or of sugar. The sufferer himself will occasionally attribute his attack to some indiscretion, such as an excess of ale, or champagne. It frequently ensues on some severe exertion, *e.g.* a long walk, carrying heavy weights, &c. It is undoubtedly excited by the combined influence of wet and cold, and especially by injudiciously 'cooling' or sitting in a draught, after a violent perspiration.

Pathology.—Not understood. It is a myalgia and is accompanied by swelling of the muscles with tension of their containing fasciæ. There is never any secondary cardiac

¹ See Dr. A. E. Garrod, *Treatise on Rheumatism*.

affection, unless the disorder be an accompaniment of acute rheumatism.

Symptoms.—The patient complains of severe lumbar pains of a stabbing or cutting character, which are worse, or often only present, on contraction of the erector spinæ group of muscles. They are therefore specially pronounced on rising to the erect posture after bending ; or on sudden movement in bed or in the chair. Occasionally spasmodic pains occur even when the patient is lying still on his back.

The disease is attended by a slight rise in temperature ; the bowels are constipated, the breath is usually foul, and the tongue thickly coated. The urine is also characteristic ; it is scanty, turbid, high coloured, of increased specific gravity, and loaded with lithates. Occasionally it contains sugar, but any slight reduction in the sulphate of copper test must not necessarily be attributed to sugar ; it may be produced entirely by the excess of lithates.

The painful condition lasts from four to five days, and leaves the patient feeble and exhausted.

Prognosis.—Good. Although the disease is painful, it is never dangerous.

Treatment.—1. RELIEVE THE PAIN.—This may be done by several local applications. We may order hot fomentations, with turpentine, or with tincture of opium sprinkled on flannel. Hot salt or hot bran bags are also excellent. Or we may apply a blister, or order liniments containing ammonia, mustard, or other rubefacient. An excellent embrocation is one composed of equal parts of belladonna and chloroform liniments. Frequently relief is obtained by a hot bath, which promotes diuresis and relieves the muscular tension. We have, however, found acupuncture the best remedy. Plunge a stout hypodermic injection needle into the masses of both erector spinæ muscles ; at the same time, if necessary, inject a small dose of morphia solution. But the puncture alone will cure in many cases. The cause of the relief is not quite evident ; but it is probably due to the lessening of the tension in the strong posterior layer of the lumbar aponeurosis.

A dose of Dover's powder (gr. x.) may be given to procure sleep and to act on the skin.

A purge is necessary if the bowels are constipated, and it can be advantageously combined with sulphur, guaiacum, and nitrate of potash.¹

In gouty patients, iodide of potassium, bicarbonate of potash, quinine, are indicated. Do not give strong purgative medicines if the bowels act, nor to debilitated patients. The diet should be simple and light. Milk, eggs, and farinaceous foods are best. Alcohol is not advisable. During convalescence quinine tonics are required.

Pleurodynia

Symptoms.—The patient complains of pain in the muscles of the chest, which is increased by movements of this region, such as occur in deep respiration, coughing, sneezing, and the like. The pain may simulate pleurisy, but auscultation fails to detect pleuritic friction, and there is no fever.

Treatment.—Medically as in lumbago.

Wry Neck (TORTICOLLIS)

Symptoms.—Supervene after severe muscular exertion; but the onset is probably aggravated, or even excited, by sleeping in a cramped position with the neck somewhat twisted. The rotatory muscles of the head and neck are especially involved, so that movement is painful or impossible, the head being usually turned to the side opposite to the inflamed muscles, and fixed.

Treatment.—Medically as in lumbago.

RICKETS

Definition.—A constitutional disease of childhood, caused by malnutrition, characterised by fever, changes in bones, probably inflammatory, which produce deformities of limbs, trunk, and cranium.

¹ R. Sulphur : ʒij. ; Pulv : Guaiaci ʒj. ; Potass : Nit. ; Pulv : Rhei, aa ʒss. Mixed with honey ; Dose ʒj. ; t.d.

Causation.—1. **AGE.**—A disease especially of infants, from one to two years of age.

2. **SEX** has apparently no predisposing influence.

3. **CONDITION OF LIFE.**—Cases occur in all classes ; but it prevails especially amongst children of the poor, and those who obtain food which is insufficient in quantity and deficient in fat and proteids. It is said that the first children of affluent parents may show signs of rickets owing to parental inexperience, and that the last children of poor parents are liable to the disease owing to increasing poverty and distress occasioned by large families. Hygienic defects, such as impure atmosphere, insufficient sunlight to underground dwellings, and the like, are also strong factors in causation.

4. **CLIMATE.**—Damp and cold. Hence it prevails in England, especially in the large towns of the north and west counties. The disease is known as ‘*Morbus Anglicus*.’

Pathology.—Probably a purely inflammatory affection principally involving the epiphysial cartilages, the shafts as a rule escaping.

At the lines of junction of these cartilages there is a proliferation of cells, a tunnelling of the cartilage by new medullary tissue, but the arrangement of the cartilage cells occurs in irregular rows with wide interspaces of matrix ; and there is also no order in which calcification takes place in these cells : one part being more advanced than another. Consequently the lines of junction between bone and cartilage are irregular in outline, with no uniformity as regards date.

In the intramembranous formation of bones, the inflammatory nature of the disease produces local thickenings and nodes, especially at those points where growth is most active. There is the same active cell proliferation, and the same irregularity and delay in the deposit of lime salts.

The liver is enlarged, owing to increase of its interstitial tissue ; but the organ is not altered in shape, and it is smooth. Similarly the spleen and the lymph glands are increased in size from hypertrophy of the trabecular tissue.

The blood is said to contain uric acid.

Symptoms.—The principal symptoms consist of the bendings and swellings of bones, which are described further on. There are, however, certain well-marked symptoms which supervene before bone changes become sufficiently definite. These symptoms have been called 'premonitory' by some; but they appear to be common to all cases, and really mark the commencement of the disease. Thus the child suffers for some little time from disorder of the respiratory and of the digestive tracts: it may be bronchitis, laryngismus stridulus, with vomiting, and diarrhoea accompanied by offensive stools. He is very peevish, fractious, and restless, especially at nights. He kicks the bed-clothes off. The skin at times is bathed with perspiration, notably the head and brow. Then possibly something happens to the child, such as a blow or a fall, and medical advice is sought, when other symptoms are found which have not previously been observed. The mother, however, generally attributes the whole subsequent ailment to the injury.

The long bones are bent and distorted, this being most marked at the junctions of the distal epiphyses with their shafts. In addition, the natural curves of bones are exaggerated. These curvatures are seen immediately above the ankle, the wrist, and subsequently above the knee, the elbow, and other joints: the epiphyses themselves being painful and swollen. The sternal ends of the ribs are bulbous, especially on their pleural aspects (rachitic rosary); the bones of the skull are soft and tender, the ordinary prominences are more marked. Thus we find bosses, more or less tender, on the frontal and parietal bones, tender and enlarged ridges at the sutures. There is also a tendency for the fontanelles to gape, or to remain open longer than normally. The face is relatively small compared to the head, though the forehead is square and massive.

Nor are these phenomena limited to the long bones. Similar tenderness may be found in the vertebral column, which not infrequently is irregularly curved and bent. The pelvic bones are misshapen, the resulting deformity being characteristic, with beak-shaped pubes, and triangular cavity,

and bulging forward of the sacrum so that the inlet becomes reniform (rachitic pelvis). The thorax is also deformed, the sternum being thrown forward owing to the falling in of the ribs immediately external to the cartilages, thus forming a shallow but well-marked groove (pigeon breast).

Tenderness also affects the voluntary muscles generally, so that the child cries suddenly when touched, or when it is washed or fondled. The skin, which is often fair and smooth, is generally bathed in perspiration during sleep, this being specially observable in the scalp, but probably occurs in equal amount in other localities. The child suffers from acidity and indigestion; the bowels are irregular, and the motions very offensive. Dentition is delayed and irregular. The liver and spleen are enlarged. The urine contains an excess of phosphates and, occasionally, free uric acid. All these symptoms are accompanied by fever, often of a hectic type. The temperature may rise as high as 103° or 104° ; but it should be remembered that anxiety does not attach to a pyrexia of this degree in a child, as it would do in an adult.

When the disease is well established there is a typical configuration and appearance belonging to the little patient. He is dwarfed; he has a large head and face, presenting well-marked eminences or 'bumps'; he is old looking for his years; he is pot-bellied; his limbs are shortened and distorted, his joints large and loose; he waddles in his gait. Yet he is often precocious, and possibly has the keenest intellect of all the children in the family; or he may become preternaturally active and muscular, if not exactly athletic.

The disease may be arrested at this point, the distortions, however, remaining during life, unless modified by suitable treatment. On the other hand, the disease may terminate in death, immediately caused by diarrhœa, bronchitis, convulsions, or other complication.

Diagnosis. — (*a*) FROM SYPHILIS. — In this disease the bony affections are commonly found before six months; and they not infrequently involve the shafts of long bones and the phalanges. There is also a history of snuffles.

(b) FROM HYDROCEPHALUS.—Here the limbs are not deformed ; nor is there the marked pain and fever of rickets. A rickety child, however, is often hydrocephalic.

(c) MOLLITIES OSSIUM.—A disease of adult life, in which there is no ossification of bones and no effort at repair.

Prognosis.—Favourable. The disease can be arrested ; although deformities are apt to be permanent.

Treatment.—Remove the child from unhealthy surroundings, and give it the advantages of fresh air and sunshine. It should be warmly clad ; it should be kept scrupulously clean, have regular hours of sleep, and its dietary should be nutritious and digestive. In short, the main lines of treatment are such as should be followed in all delicate and improperly nourished children. If an infant, it would be as well to obtain a healthy foster-mother, or, failing this, to feed the child on cow's milk diluted with one third water and a little sugar added. If dentition has commenced we may order extracts of animal food, beef-tea, mutton or chicken broths, or the juice of raw meat. Farinaceous puddings, oatmeal, and other starchy foods are to be allowed sparingly only. Care must be taken not to overload the stomach, nor to give any food which appears to cause indigestion.

As a medicine cod-liver oil appears of greatest value. It may be given in the form of emulsion, or mixed with milk. A teaspoonful three times a day is enough to begin with.

Beef-suet is an admirable substitute for the oil. It can be made palatable by being boiled in milk.

Lime salts appear to exert little effect unless in combination with iron, as in Parrish's food. The syrups of the iodide or the phosphate of iron are also useful.

The distortion of limbs and vertebral column must be remedied by mechanical or by surgical measures. We may recommend gymnastics if the child be old enough, also passive movements of limbs, shampooings, and the like. Anything requiring force or laboured exertion, however, should be deprecated.

In an infant an excellent plan is to allow it to lie on a pillow or bed for an hour or so daily, when it will probably

use its limbs vigorously. But all attempts to walk or to creep are to be discountenanced.

So long, however, as fever and pain are present, all movements of any kind are inadvisable.

Regulate the bowels with slight laxatives (castor oil best). When there is diarrhœa with offensive stools, give alkalies and carminatives.¹

DIABETES MELLITUS

Definition.—A disease characterised by the excretion of a large quantity of urine of high specific gravity containing glucose ; and by great thirst with progressive emaciation.

Causation.—**AGE.**—Most frequent between twenty-five and forty : but it occurs at all ages.

SEX.—Males suffer most, in the proportion of about two to one.

NATIONALITY.—It is a disease especially prevalent amongst the Jews and the Hindoos.

HEREDITY.—Often a strongly marked factor.

NERVOUS DISORDERS.—It often is preceded by organic disease of the brain or medulla ; by injury to the central nervous system ; and by mental strain and anxiety. A dread of the disease may even induce it in this way.

DIATHESIS.—Strumous and gouty diatheses act as pre-disposing causes.

OTHER DISEASES.—It may supervene on, or be secondary to, diseases, whether functional or organic, of the liver (hepatic diabetes), the pancreas, or the brain.

In many cases, however, the causation is doubtful, and, indeed, not evident at all.

Pathology.—Still obscure. The body is generally emaciated and free from fat. The kidneys are increased in size owing to their activity. The lumen of the tubules is enlarged, the epithelium swollen and granular, and the individual cells appear to absorb logwood and other staining

¹ R₂. Magnes: Carb: gr. ij.; Pulv: Cretæ Aromat: gr. ij.; Sp: Ammon: Aromat: mij.; Aquæ Pimentæ ad ʒj. Miscæ.

reagents with difficulty, probably owing to the presence of fat. Hadden and others have found fat in the blood (lipæmia), and also a 'cirrhotic' condition of pancreas. Dickinson has described certain lesions in the brain and central nervous system, especially dilatation of the blood-vessels, extravasations into the perivascular spaces, with thickenings of the vascular sheaths, and degeneration of 'circumjacent nervous substance.' In the cord he found dilatation of the central canal. Bernard's physiological experiment, in which he produced glycosuria by irritating the floor of the fourth ventricle, has apparently been the centre round which pathological research has worked; and in view of the close connection or communication between the medulla and the highest cervical sympathetic ganglion, there is reason to suppose that irritation of the medulla and base of brain may be by this route transmitted to the hepatic vessels, causing hyperæmia of the liver and consequent increased glycogenic function.

Symptoms.—The onset is usually gradual and insidious. Cases have even been admitted to hospital in the comatose state of the last stage, in whom no previous suspicion of disease had been entertained.

Usually, however, abnormal conditions of urine attract the patient's attention. He passes an unusual quantity. Micturition is frequent during the day; and he has to rise from his bed two or three times during the night. Or he may tell us that his urine has a peculiar sweet odour, comparable to the scent of sweet-briar, or of apples, or of new-mown hay; that it is very frothy, and attracts flies, wasps, or other insects to the utensil, and that the orifice of the urethra (or the vulva in a female) is sticky and ex-coriated.

These symptoms usually compel him to seek medical advice, when it is found that he is passing urine in large quantities (ten to fifteen pints or more a day), of high specific gravity (1030-45), of light pale green tint, and containing large quantities of glucose. The sugar in the urine acts as an irritant to the bladder and thus causes frequent micturition, the

bladder being by no means full before each evacuation. It is as well, also, to remember that cases occur in which the specific gravity of the urine is little, if at all, raised. The amount of sugar varies; about ten per cent. is the usual proportionate quantity in each sample, and fifteen to twenty-five ounces the gross daily weight. But these figures will vary considerably according to the patient's diet. The amount of urea excreted *per diem* is also in excess of the normal; but in each sample of urine it is proportionately less. Still, excess of urea may account for a very high specific gravity in those cases where, on analysis, the amount of glucose is insufficient to cause it.

The blood also contains an appreciable amount of glucose.

It is also found that the patient has an insatiable appetite, although he steadily loses weight; that he suffers intensely from thirst, especially during the night; that he has constipation, the stools being dry and scanty; that his tongue is abnormally red, clean, and fissured; and that the temperature is subnormal.

As the disease progresses, the skin becomes harsh and furfuraceous, almost all the fluid which he drinks being carried off by the kidneys; the teeth fall out; the face becomes careworn and preternaturally aged and wrinkled; the pulse is small, feeble, and quickened; emaciation proceeds apace; he complains of sinking feelings in the epigastrium, of aching in the loins, and he has a tendency to faintings.

The mental attitude is also noteworthy. He is listless, despondent, or morose, and disinclined to exertion, both bodily and mentally. The knee-jerks are diminished or absent; sexual power is lost, although seminal emissions occur from time to time without voluptuous sensations. Although the disease is mostly a progressive one towards a fatal ending, it may be abated, or even temporarily arrested. Death is usually due to exhaustion, to coma—which may come on suddenly and unexpectedly—to pulmonary phthisis, or to gangrene.

Diagnosis. A persistent polyuria, with glycosuria, a urine of high specific gravity, accompanied by thirst, an enormous appetite, and emaciation—these symptoms can be due to no

other disease than diabetes. We may confound it with temporary or acquired glycosuria, found in gouty subjects and high-living patients ; but the symptoms abate with treatment, the glycosuria being due to abnormal hepatic stimulation. A temporary glycosuria, however, may become permanent by continuous ingestion of an excess of carbo-hydrates.

Prognosis.—Always unfavourable. Recovery is rare ; but, with care and a rigid dietary, life may be prolonged for eight or ten years. Death, however, has been known to occur in a few weeks from the recognition of the disease. The younger the patient the more gloomy the outlook. About the twentieth year it is rapidly fatal.

Complications.—1. **SKIN.**—The patient is liable to many forms of cutaneous disorder. The most frequent are boils, carbuncles, eczema, xanthoma, indurated and inflamed acne. Eczema and fissures about the prepuce, glans penis, or labia majora are the direct result of the irritation caused by the sugary urine. General anasarca, due to feeble action of the heart, and gangrene often occur in the last stages.

2. **EYES.**—Diabetics are subject to cataract. It may affect one lens or both, and may occur in quite young subjects.

3. **RESPIRATORY TRACT.**—Tuberculous phthisis is the most frequent complication, large caseous deposits, followed by excavations, occurring in the apices and other parts of the lungs. A chronic ‘smouldering’ pneumonia occasionally attacks the bases, the tubercular bacillus becoming subsequently engrafted (chronic pneumonic phthisis).

4. **DIGESTIVE TRACT.**—Profuse diarrhœa, especially towards the end of the disease.

Treatment.—1. **REGULATE THE DIETARY** so as to allow as small amount of starch and sugar (carbo-hydrates) as possible.

Thus, (*a*) *the patient may eat* beef, mutton, pork, ham, bacon, poultry, game, fish, eggs, cheese, butter, cream, green vegetables, asparagus, vegetable marrow, mushrooms, lettuce, endive, tomatoes, radish, colery, vinegar, oil, pickles, jellies and blanchmange (not sweetened), nuts, olives, mayonnaise, and the almond, bran, or gluten, or soya breads.

(*a*) *He may drink* tea, coffee, dry sherry, claret, Burgundy,

brandy (if good), whisky. Occasionally bitter ale may be allowed, or lager beer ; but the less alcohol the better.

(b) *He must not eat* sugar, wheaten bread and biscuits, rice, arrowroot, cornflour, oatmeal, sago, tapioca, macaroni, potatoes, peas, carrots, parsnips, beetroot, pastry, and fruit.

(b) *He must not drink* sweet ales, porter or stout, cider, champagne, port wine (unless sparingly), liqueurs. (Modified, after Pavy.)

2. ALLEVIATE DISTRESSING SYMPTOMS.—Thirst appears to be best relieved by opium. We may give as much as six to eight grains *per diem*, in half-grain doses. Ice, lemonade, without sugar, and acidulated drinks may be freely allowed.

Hunger can only be appeased by food ; but cod-liver oil is an excellent adjunct. Begin with small doses (a teaspoonful) and gradually increase them.

Relieve constipation by vegetable aperients (rhubarb, cascara sagrada, castor oil, jalap), but avoid mercurials. Eczema, boils, and other skin diseases are to be treated on general principles. It is as well not to incise a boil or carbuncle, owing to the tendency to the formation of an unhealthy wound. The external genital organs should be kept scrupulously clean by frequent washings ; excoriations and chappings being obviated by vaseline, lanolin, or other greasy application.

3. WARD OFF COMPLICATIONS. — A warm yet bracing climate is desirable, if phthisis be threatened. Watch for any tendency to coma, which may come on suddenly, but is often preceded by irritability of temper and restlessness. Dickinson has advocated intra-venous injection of a saline solution, consisting of chlorides of sodium and potassium, sulphate of soda, sulphate of potash, and bicarbonate of soda.¹

4. ARREST THE EXCRETION OF SUGAR.—Opium and its alkaloids afford the best chance. Begin with small doses and gradually increase them up to four grains or more *per diem*. Dr. Pavy recommends codeia (gr. $\frac{1}{2}$ t.d.). The skim-milk treatment—six pints a day—has occasionally afforded good results.

¹ See *Brit. Med. Journal*, March 1890.

5. GENERAL RULES.—The body should be warmly clad. Hot and vapour baths may be taken with a view to increase the skin's action. Blisters to the neck, although advocated by some authorities, have a tendency to form gangrenous sloughs. Operations for cataract are not usually successful, and should therefore be deprecated. The patient should avoid fatigue and exposure, and try to follow rules which are ordinarily conducive to general health.

It will be frequently observed that patients have a tendency to over-ride orders, and that treatment is therefore difficult to carry out. The craving for bread, for example, is often difficult to control, and it will be found at times that treatment is more irksome than the disease. In certain cases, indeed, when the amount of glycosuria is stationary, we may even allow a small quantity of brown or toasted bread.

DIABETES INSIPIDUS

Definition.—A disorder characterised by the excretion of an abnormal amount of clear urine, containing no sugar, and not related with obvious structural disease of the kidneys.

Causation.—1. AGE AND SEX.—Adult males suffer most frequently, owing, possibly, to alcoholic excesses.

2. HEREDITY.—It is said to 'run in families'; the disease itself not being inherited, so much as certain predisposing nervous conditions.

3. NEUROSES.—Such as shock, hysteria, injury to brain, syphilitic disease of brain.

4. GENERAL DISEASES.—It appears to be consequent on continued and other specific diseases, and tubercle. The causation is, however, mostly obscure; and it appears *per se* in many cases to be a symptom rather than a disease.

Pathology.—The kidneys may or may not present pathological changes. Usually, the renal tubules are distended and lined with swollen granular epithelium. The disease appears to be essentially a functional disorder affecting the vaso-motor system.

Symptoms.—In many points they bear a close resemblance to those of Diabetes Mellitus. Thus, there is thirst, which is proportionate to the amount of urine which is voided, with emaciation and dry skin. The urine is in great excess—thirty pints or more *per diem*; it is clear, limpid, its specific gravity about 1005, but it contains no sugar. In addition, the patient often complains of lumbar pains; he is anæmic and feeble, and possibly irritable or peevish. On the other hand, he often enjoys good health; the constant desire to micturate being the only distressing symptom. In some forms the amount of urinary solids is decreased, whilst in others the urea is in large excess.

Not infrequently the disease is a precursor of true saccharine diabetes.

Prognosis.—Less unfavourable than diabetes mellitus; but if it occur after shock, or some prostrating illness, it must be regarded as a serious disease. The chief danger appears to arise from emaciation and exhaustion.

Diagnosis.—FROM CHRONIC BRIGHT'S DISEASE.—In diabetes insipidus there is no albuminuria, and no evidence of cardio-vascular derangements.

FROM CHRONIC ALCOHOLIC EXCESS.—Here we should be guided by the alcoholic history, the odour of the patient's breath, gastro-hepatic disturbances, and muscular tremors.

Treatment.—Rest, freedom from worry, anxiety, and excitement. Avoid drinking an excess of any fluid. Ergot, opium, and valerian afford the best results, so far as drugs are concerned.

SPECIFIC INFECTIOUS DISEASES

FEVER (PYREXIA)

Definition.—An abnormal condition in which there is elevation of temperature above the healthy range.

Causation.—Still a matter of speculation and theory. It is obvious that in health a certain amount of heat is generated in the body; and it is equally obvious that a corresponding amount of heat is lost by the skin, muscles, glands, and nerve tissues. These amounts of income and expenditure should exactly balance, and so maintain an equable normal temperature of the body, with only a slight limit of variation. Any disturbance, from whatever cause, between the adjustment of heat, developed in the interior, and the amount lost by respiration, radiation, and the emunctories, constitutes the condition known as *fever*. The actual generation of heat is thought to be under the control and guidance of a supposed 'calorific centre' situated in the middle of the external surface of the brain. If this be so, any interference with the nerve fibres passing from this centre will produce abnormal conditions of temperature. But any alteration in the calorific centre itself may induce tissue changes, either by increasing both heat production and heat discharge (the two functions being maintained at a higher level than is normal), or by loss of its inhibitory or controlling power, the heat centre itself being paralysed.

On the other hand, pyrexia is thought to be due to the action of contaminated blood directly on the tissues, causing chemical changes in them.

Whichever view be correct, it is certain, from cases recorded by Hale White and others, that fever has attended lesions of the central nervous system, whether they be mechanical injuries, or pathological changes such as tumours, hæmorrhage, sclerosis, and the like.

It also is certain that fever occurs in blood-poisoning. This may be brought about by the circulation of the products of microbes, or by the microbes themselves, but these latter are not essential. Or the blood itself, by certain changes and metamorphoses, and the absorption of the products thereof, may be directly responsible for pyrexia. This is seen in cases of embolism, thrombosis, and extravasation.

Symptoms.—Usually three stages are recognised, viz. (1) Cold or rigor, (2) Acme, (3) Termination. In the first stage the skin is cold, and pale, or livid; this is due to spasm of the superficial arterioles. The internal temperature, however, is increased, as the actual production of heat is augmented, whilst the surface loss is diminished. This stage usually lasts a few hours, or even a much shorter time.

In the second stage both production and loss of heat are increased. There is an increased consumption of fuel (O) and an increased discharge of the products of combustion (CO_2), and all at the expense of the nitrogenous tissues generally. There is also a marked increase of the excretion of urea. Carbonic acid is, however, only increased relatively, and not actually in each expiration.

This stage may continue for days or even weeks.

The third stage, or termination, is characterised by (a) Crisis, with a sudden fall of temperature to the normal line or even below it, accompanied by a profuse perspiration, or by diarrhœa, salivation, or other signs of glandular activity, or by more or less pronounced delirium. (b) Lysis, in which the fall of temperature and the improvement in symptoms are gradual.

A more detailed account of the symptoms which accompany fever is, however, necessary, and, with a view to some regularity of description, we have grouped the different symptoms into systematic headings.

(1) *Gastro-intestinal*.—The tongue is dry and foul ; there is loss of appetite ; thirst is always a marked symptom : the bowels are usually constipated, but there may be, on the other hand, diarrhœa and vomiting. As a result of the excessive loss of nutrition, there is more or less general wasting, especially affecting the adipose, muscular and glandular tissues.

(2) *Respiratory*.—The respirations are shallow and quick (30 to 50) ; but the increased frequency is not in relation with the heightened temperature, as is the pulse. Frequently the febrile condition is attended by hypostatic congestion at the bases of the lungs, this being due partly to the shallow respirations and partly to the feeble cardiac systole.

(3) *Circulatory*.—The pulse is increased in frequency, and bears a corresponding ratio to the increased temperature. It is usually full and bounding ; its tension is also increased as a rule. Exceptions to this latter rule occur in weakly subjects, and in the critical sweating stage, when its tension is low ; it may then even be dicrotous. The red corpuscles are diminished in number, and the blood is further impoverished in its albuminous constituents.

(4) *Urinary*.—The urine is diminished in quantity ; its colour is darker from disintegration of the blood cells ; it is acid, and the specific gravity is increased, owing to the aggregate of its solid constituents being in excess. The amount of urea excreted is about twice the normal quantity, and there is almost a similar ratio of excess of uric acid. The chlorides, however, are diminished.

(5) *Cutaneous*.—The skin is usually dry, hot, and pungent, or, if there be much capillary tension, it is cold and dusky. Exceptions, however, are seen in pyæmia and acute rheumatism, which are accompanied by profuse perspirations.

(6) *Nervous*.—Amongst the nervous symptoms must be mentioned rigors, headaches, general pains, with twitchings of muscles, or even convulsions. Various types of delirium and stupor are common, their amount and severity varying in different fevers.

Pathology.—After death the blood is found imperfectly coagulated ; the heart and vessels are usually deeply stained

by it. The spleen is swollen and soft ; the kidneys and liver are enlarged, and present signs of parenchymatous degeneration. The muscles also are degenerated and soft. In typhoid fever waxy degeneration of the muscles, especially of the adductors, is frequent. Further, special fevers appear to expend their virulence in certain organs or tissues, and so produce characteristic lesions. Thus, the small intestine is especially involved in typhoid fever, the soft palate and pharynx in scarlet fever, the meninges in epidemic cerebro-spinal fever.

TREATMENT.—No definite rules can be laid down. Our treatment depends on the disease of which pyrexia is a symptom.

VARIETIES OR TYPES OF FEVER

1. **The Typhoid State.**—This term is used to indicate the stupor which attends the graver kinds of fever. The patient is prostrate ; he has marked muscular debility ; he lies on his back and tends to sink low down in the bed ; the extremities are cold ; he has muscular vibrations or twitchings (*subsultus tendinum*) ; his fingers pick at the bed clothes, or search for imaginary objects ; the tongue is dry, fissured, and contracted ; sordes collect on the teeth and lips ; the bowels are irregular ; his evacuations, exhalations, and skin are offensive ; his respirations are shallow and quickened ; his pulse-rate is accelerated (120 to 140), its quality is feeble, often imperceptible or dirotous ; the cardiac systole is almost inaudible ; cutaneous sensibility is impaired ; bedsores form ; his special senses are blunted ; he has a dull, staring expression of countenance, with an absence of real sleep (coma vigil) ; he is delirious, yet can be roused from time to time to give fairly intelligible answers. Towards the end he passes his evacuations unconsciously, but there may be retention of urine, and he eventually sinks into profound coma which ends fatally. This condition is due to the defective elimination of the effects of active tissue changes observed in fever.

Examples.—Typhus fever, erysipelas, and the last stages of cirrhosis and acute yellow atrophy of the liver.

2. **Hectic Fever** is chiefly characterised by its gradual onset, its chronicity, and its distinctly remittent type. It is attended by perspirations, a rapid pulse of low tension, a well-marked, circumscribed flush on the cheeks, with emaciation, and usually diarrhœa.

It is due to the circulation in the blood of some specific poison producing a fever which does not run a definite course, and usually accompanies those diseases which are associated with prolonged suppuration.

Examples.—Phthisis, dysentery, caries of bone.

3. **Adynamic Fever.**—This form of fever is characterised by intense prostration of strength (asthenia) without any great or corresponding rise of temperature.

Example.—Diphtheria.

4. **Continuous Fever** is a term applied to those forms of pyrexia which persist for some days at a fixed level without any marked variation of rise or fall.

Examples.—Sunstroke ; the febrility attending overwork and consequent exhaustion.

5. **Remittent Fever** is characterised by successive exacerbations alternating with remissions ; but the remissions of temperature do not fall to the normal level.

Examples.—Remittent, and some of the ill-defined fevers of Oriental countries.

6. **Intermittent Fever** is somewhat similar to the above ; but the remission of temperature actually reaches the normal line. The intermission is thus complete.

Example.—Quotidian ague.

7. **Relapsing Fever** is that form of febrility which occurs in outbursts, with periods, more or less prolonged, of complete apyrexia between.

Example.—Relapsing or famine fever.

8. **Specific Fever.**—This name is given to those diseases which are due to the circulation of some specific organism in the blood, where it increases and flourishes so as to be capable of propagation from one individual to another (*see* SPECIFIC FEVERS).

Examples.—Small-pox, scarlet fever, cholera.

9. **Inflammatory Fever** is a term used to express the febrile condition consequent on some local inflammation or injury.

Examples.—Simple bronchitis, synovitis, abscess.

10. **Malignant Fever** is characterised by its suddenness and its severity. It is always of a low type and is usually rapidly fatal.

Examples.—Malignant small-pox, and typhus, especially in severe epidemics (*Typhus Siderans*).

THERMOMETRY

Some precautions and care are necessary in taking a thermometrical observation. The thermometer should be adjusted by shaking the index down to 95° , but of course not so low as the bulb; the index itself should consist of a column isolated from the rest of the mercury; it should not be broken up into two or more fragments. With these preliminary precautions there can be no error in a high observation, as heat alone will cause the index to rise; there may, however, be an incorrect low observation in cases where the thermometer is not properly laid.

For all practical purposes the axilla is the best site to apply the instrument. The axilla should be previously cleansed from perspiration, the bulb of the thermometer should be pushed upwards towards the summit; it should touch the skin on all sides, and no garment or bandage should be allowed to intervene. In muscular men the pectoral fold may hold the stem of the instrument whilst the bulb does not touch the skin, and hence an inaccurate observation is made.

Other sites for thermometrical observations are the mouth (sublingual), the anus, the vagina, and the groin. The mouth is objectionable in young children owing to the danger of their biting and breaking the instrument. Adult patients also rebel on the ground of uncleanness. A rectal observation may be rendered unreliable by the impact of dry faeces, when the reading would be too low, or by the advent of diarrhoea, when

too high an observation might be recorded. Accurate results may be obtained in infants by laying the thermometer in the groin and then flexing the thigh on the abdomen. It is important to remember that diseases in children cause much higher ranges of temperature than they do in adults, and that a reading of 103° or 104° may be caused by slight ailments which would probably not raise the thermometer above 100° in men.

The following table, modified from Finlayson's 'Clinical Manual,' gives the nomenclature of terms in general use in clinical thermometry :

Above 105° F.	Hyperpyretic	{	In typhus fever, acute rheumatism, lesions of the nervous system, &c.
From 104° to 105° F. .	Highly febrile	{	In typhoid fever, pneumonia, pericarditis, &c.
From 102° to 103° F. .	{ Moderately febrile	{	In bronchitis and catarrhal affections generally.
From 99.5° to 101° F. .	Sub-febrile	.	In slight ailments.
98.6° F.	NORMAL.		
From 97° to 98° F. . .	Sub-normal	.	In crises after any acute fever.
From 95° to 96.5° F. .	Collapse	.	In cholera and shock.

In determining the intensity of a fever the hand alone is insufficient, although it may detect certain important signs, such as pungency of skin in pneumonia, or the cold, clammy state of collapse ; a patient's own sensations are equally untrustworthy. The thermometer must be used. The normal temperature for an adult is 98.6° Fahr., but variations of half a degree are not incompatible with health. A full meal, muscular exercise, or even an exciting argument, will raise the temperature above normal. On the other hand, alcohol will reduce the temperature.

[In the description of all febrile states the thermometrical observations are made according to the Fahrenheit scale.]

SPECIFIC FEVERS

Definition.—Fever which is dependent for its origin and propagation on some specific poison, which produces in each case special and distinctly characteristic phenomena.

Characters.—They all possess many features in common.

1. The poisonous matter, to which a given specific disease is due, when it is received into the system, multiplies to an extent which is beyond our powers of computation. It may be compared to seed which, introduced into the soil, germinates and produces fruit, having known appearances and characteristics. Thus, the poison of syphilis produces syphilis and only syphilis, and the poison of scarlet fever gives rise to scarlet fever and nothing else, by whomsoever received. And, as in the vegetable world some seeds under unfavourable surroundings may never germinate, or grow only to an immature stage and then wither and die ; so in specific diseases, we may meet with abortive attacks : and it is fair also to presume that in some cases the poison, although introduced into the system, gives rise to no signs whatever, apparently from some condition in the animal tissues which is inimical to its propagation.

2. There is, therefore, in each specific fever a spore or fungus, a bacillus or microbe, communicable from the sick to the healthy, and which, in its development and growth, 'breeds true.'

3. The specific poisons may be discharged by the breath (typhus fever), by the skin (scarlet fever), or by the bowels

(typhoid fever), or by all three channels, and they likewise may be received by the victim through similar, and indeed, in some instances, through all routes.

4. All specific fevers have latent periods, or periods of incubation, during which the poison, although admitted to the blood, is maturing, but produces no immediate effect.

5. The poison, having entered the circulation under favourable circumstances, gives rise to certain febrile symptoms, in most cases accompanied by a cutaneous eruption or rash. The rash when present is diagnostic.

6. The decline of the fever is marked in some instances by a sudden fall in the temperature and amelioration of symptoms (crisis), in others by a gradual decline (lysis).

7. A specific fever confers on the individual a temporary immunity from a second attack. On this feature are based the principles of the researches of Koch, Pasteur, and others. It must not be forgotten, however, that this does not hold good in every specific fever ; and, moreover, some people, by peculiar idiosyncrasy, seem particularly prone to second and even third or fourth attacks.

8. Some specific fevers appear to be peculiar to mankind, and even some of them peculiar to, or exhibit a preference for, certain races of mankind. Others, again, appear to be special to the lower animals, and whilst not necessarily so severely fatal to them, they possess most malignant qualities when acquired by man. It is probable that the reverse of this is also true.

9. Some are distinctly epidemic in character, visiting continents, or localities, or communities, at intervals of short or long duration. Generally these fevers are highly infectious. Others are endemic, appearing to rest permanently with a nation or in a locality. As a rule these are less infectious.

Those fevers which visit us in epidemic form may be conveyed by the winds, but often through some agency quite undiscovered. The death-rate which they cause is at first heavy, as these epidemic fevers select for their first victims the weakly members of a community. The death ratio falls as the epidemic gradually exhausts itself. During their

prevalence in epidemic form they stamp other ailments with some of their peculiarities. Thus, diarrhœa is often urgent and suspicious during outbreaks of cholera ; sore throats are frequent during epidemics of scarlet fever.

10. During the course of a given specific fever there does not appear to be room in the blood for any other specific disease. It would seem to engage all the vital properties of the sufferer, and no matter what further exposure to any other fever may take place, it is not received into the system. Exceptions to this rule, however, have been recorded.

Treatment.—It may be stated that in specific fevers we know of no drug or remedy which will arrest or shorten their course. Exception to this rule must be made in the case of quinine (for ague) and mercury (if syphilis is to be regarded as a specific fever). Further, recent researches point to the possibility of many specific fevers being stopped at their onset, or modified in their course, by inoculation of diluted virus.

The general treatment of a specific fever may, therefore, be said to consist of nursing and care rather than drugging. Conduct your patient through his fever, and use only medicinal remedies for complications and urgent symptoms.

The room should be large, airy, and, if possible, at the top of the house. Remove all carpets, curtains, and unnecessary furniture, and clothes. See that there is a free ventilation by windows and chimney.

The bedstead should be an iron one, of single size, and placed away from the walls. Remove and at once disinfect foul bed-linen, bed-gown, and all towels or handkerchiefs used by the patient. Drinking and other vessels and all utensils used by the patient should be kept entirely separate and disinfected from time to time after use.

No visitors should be allowed beyond his doctor, his nurses, and perhaps a near relative.

His attendants should not be any closer to the patient than is necessary. Their hands and persons should be kept scrupulously clean, and, if possible, their garments changed before coming in contact with other people. A nurse had

better be selected who has already suffered from the fever in question. The patient cannot be regarded as free from risk of conveying contagion until every particle of desquamated skin or of scab has fallen from his body, and until he himself has been thoroughly cleansed by some disinfecting bath.

The room subsequently should be effectually disinfected by fumigation, and then, if possible, re-papered and painted. The best method of disinfecting a room is by burning sulphur on a pan of live coals, and closing all doors, windows, and crevices. Bedding should be treated to dry heat of 212° Fahr.

SMALL-POX (VARIOLA)

Definition.—A specific, contagious, eruptive fever, the eruption passing through the stages of pimple, vesicle, pustule, and scab.

Causation.—1. **PREDISPOSING CAUSES.**—All ages and both sexes are liable to the disease; it is more prevalent in summer, and in close, unventilated places. Tramps and vagabonds appear specially susceptible. The most important predisposing cause, however, is want of vaccination.

2. **EXCITING CAUSE.**—A specific virus given off from the breath, skin, and emunctories. The disease can be carried from one person to another by an attendant or medical man. The infective distance is probably not greater than two yards.

Incubative Period.—Twelve to fourteen days.

Symptoms.—**PRIMARY FEVER.**—Sudden and severe, the temperature rising at once to 100° , and 102° – 104° in the first three days. It is marked by a characteristic pain in the back, the patient feeling as though he had been beaten by a heavy stick. He also suffers from general malaise, headache, vomiting, and perspiration. This stage lasts about three days.

ERUPTIVE STAGE.—The eruption may be preceded by a scarlet rash or blush, which may be generally diffused, or local only. The true rash appears about the end of the third

day, first showing itself on the wrists, chin, corners of the mouth, and the neck. It then extends to the face, trunk, and upper extremities, and finally to the lower limbs, being usually a day later in the legs in all its stages as compared with the upper parts of the body. The eruption at first is papular (pimples), and very hard or 'shotty' to the touch. Three days later the papules become vesicular, each vesicle being surrounded by a red areola and filled with opaque fluid. As the vesicle increases in size and is distended with its contents, it becomes depressed in its centre (umbilication), owing to a bridge of cellular tissue attached from the summit to the true skin beneath. At this period there is generally marked tumefaction of skin.

Two days later (8th of fever), the bridge ruptures, the vesicles become pustular and exude their contents, giving rise to a disgusting smell.

SECONDARY FEVER.—The temperature, which had fallen on the onset of eruption to 99° or 100° , now again rises to 103° or 104° , which usually lasts about two days, when the pustules fall in and maturation takes place (14th day of fever, 11th of eruption). The scabs which form then fall off, and continue doing so until about the end of the third week (21st day), leaving a red pitted scar with numerous minor depressions in its centre.

But the eruption is not entirely limited to the skin, since similar phenomena may be observed in the mouth, attended by salivation, and found, *post mortem*, in the pharynx, œsophagus, and air passages. In addition there are symptoms of nervous prostration (subsultus and delirium), tremors and loss of control over the sphincters.

Varieties of Small-pox.—1. **DISCRETE.**—As described above.

2. **CONFLUENT.**—The pustules run together or coalesce; the earlier symptoms are more severe, the remission in fever either does not take place, or, if it occur, it commences earlier, and is less marked and of shorter duration; delirium, convulsions, and the 'typhoid state' are common.

3. **VARIOLA CORYMBOSA.**—A somewhat fanciful name applied to that form in which the pustules are arranged in currant-like clusters or groups. (Not important.)

very severe, and that the extent to which the disease is modified may vary according to the susceptibility of the individual.

6. INOCULATED SMALL-POX.—Practised in Eastern countries (China, Japan) by introducing the virus under the skin, or by the disgusting method of placing a crust from a variolous eruption into an abraded nostril. There is an incubative period of one day only, when a pimple forms at the site of inoculation, which becomes pustular on the eighth day. Three days later there follows a crop of papules, which run through the ordinary course. The disease thus acquired is a comparatively mild one, but it is nevertheless a centre of infection to others, who may develop the disease in its severer forms. It is penal in this country to inoculate small-pox, and can only be tolerated on board ship, or in such localities where the population is unprotected by vaccination, and where no vaccine lymph can be obtained.

Pathology.—No special appearances in any organ beyond the congestions of lungs and kidneys, common to all febrile diseases. The blood is usually uncoagulated and of somewhat tarry consistency ; the heart muscle is pale and flabby.

Diagnosis.—FROM VARICELLA.—The febrile symptoms are slight, the eruption in its early stage is not hard, and never goes on to pustulation unless irritated.

FROM MEASLES.—The eruption first appears on the forehead ; it is not shotty to the feel. Symptoms of catarrh of the respiratory tract are always present.

FROM ACNE.—In which there is no fever. Further, the sebaceous plug can be expressed and examined.

Prognosis.—The prognosis in an ordinary attack varies according to the efficiency of vaccination ; in other words, the mortality decreases in steady ratio according to the increased number and size of vaccination scars. Statistics show a death rate in round numbers as follows : unvaccinated, 50 per cent. ; vaccinated, 6 per cent. ; efficiently vaccinated, 2 per cent.

The fever is especially fatal in the extremes of age, and to Negro and Oriental races. The presence of petechiæ or of hæmorrhage anywhere should be regarded as of bad omen. The

same remark applies to much delirium. The period of secondary fever is the critical time of the disease.

Complications and Sequelæ.—1. CUTANEOUS SYSTEM.—Erysipelas, abscesses (with resultant pyæmia).

2. RESPIRATORY TRACT.—Œdema of glottis (whether or not as the result of eruption in the air passages), bronchitis, broncho-pneumonia, pleurisy.

3. DIGESTIVE TRACT.—Severe ptyalism, sloughing of mucous membrane of mouth, severe vomiting and diarrhœa.

4. PREGNANCY.—The patient usually aborts and dies. Still, cases are recorded in which the mother has survived the attack and produced a child 'at term' with small-pox scars on its skin.

5. SPECIAL ORGANS.—*Eye.*—Conjunctivitis, abscess of lachrymal duct, sloughing of cornea. *Ear.*—Otitis.

6. NERVOUS SYSTEM.—Maniacal delirium, convulsions, post-febrile palsy of limbs, aphasia, or a drawling, 'scanning' speech, as in disseminated sclerosis.

Treatment.—1. *Prophylactic.*—Vaccination may be performed with salutary results as late as the sixth or seventh day after exposure to small-pox.

2. There is no treatment special to the disease. Place the patient, if possible, in a large, well-ventilated, cool room; this is important, as experience shows that small-pox cases which are placed in tents or well-ventilated wooden huts have much less subsequent scarring. In other words, regard each pustule as requiring antiseptic surroundings, of which fresh air is the best. The diet should consist of milk, eggs, and beef-tea, given in frequent quantities in order to maintain the patient's strength. Iced drinks may be allowed. Attend to the state of the bowels; they should act daily. On the other hand, check diarrhœa and vomiting should they occur. Beyond the above, treatment should be directed to alleviate symptoms and complications. Cold lotions may be applied to the face if there be much irritation; or the face and body may be anointed with carbolised oil, cold cream, &c. With a view to prevent disfiguration, it has been recommended that the papules on the face be painted with collodion to exclude

the air, or that the vesicles be punctured with a fine needle ; even weak solution of nitrate of silver has had its advocates, but, as stated above, we have seen better results from free ventilation in tents, than in hospital wards or in private houses. Delirium and sleeplessness require the exhibition of large doses of bromide of ammonium (gr. xx.), or small doses of opium carefully given, and withdrawn if unfavourable symptoms arise. Restraint is not advisable unless absolutely necessary, nevertheless the patient should be constantly watched. Be prepared to treat immediately ophthalmia or œdema of the glottis, both of which may supervene suddenly. During convalescence from so exhausting a fever, a full diet, with some wholesome stimulant, is demanded. Tonics (cinchona, mineral acids, &c.) may also be ordered with advantage.

It is important to remember that it is a strongly infectious disease, especially to unvaccinated people, and that the danger of imparting the disease remains until the last scab has fallen.

VACCINATION

Definition.—Inoculation of cow-pox (*Vaccinia*).

Symptoms.—The virus can be introduced into the system by inoculation only, after which there is an apparent incubative period of two days. A papule, with an inflamed areola, forms on the third day at the point of inoculation ; this becomes vesicular on the sixth day, when it is distended with clear yellowish lymph and distinctly umbilicated. On the eighth day pustulation occurs, after which the crown of the pustule falls in, the contents dry up and form a scab, which eventually falls off about the twenty-first day, leaving a depressed cicatrix, which is permanent. The constitutional symptoms are as a rule insignificant. There may be a slight increase of temperature during the vesicular and pustular stages, and occasionally implication of the nearest lymphatic glands takes place.

The above symptoms may be modified. Thus, the various stages of the eruption may be retarded, or they may be accelerated and run their course in one or two days less than is usual. Again, the eruption may not pass through the ordinary

stages, but cease at the papular stage, forming an abortive form of the disease which does not necessarily give any immunity from small-pox.

Theories.—1. That it is a disease antagonistic or antidotal to small-pox. 2. That it is the same disease as small-pox, which, having passed through the cow, becomes in some measure a modified disease, and which, although comparatively inert when re-introduced into the human blood, confers immunity from an attack of true small-pox. It may safely be asserted that, given perfect vaccination, small-pox should almost be an unknown disease, and, indeed, the records of the public health of the community in the last century, and pictures and portraits of the people, abundantly testify to the prevalence of this scourge prior to Jenner's priceless discovery.

Treatment.—No treatment is usually required beyond protecting the inoculated points from pressure and injury. If there be any marked febrile disturbance, a small dose of castor oil or other laxative may be prescribed, followed by saline febrifuge (liq. ammoniæ acetatis).

Vaccination is best performed by the direct method from arm to arm. Select a healthy child (vaccinifer) of healthy parents, who has been vaccinated and in whom the vesicles are fully developed and of normal appearance. Open a vesicle on the sixth day, or not later than the seventh, else the vesicles are tending towards pustulation. When a bead of lymph has exuded, scarify the arm of the recipient near the insertion of the deltoid (the right arm is usually selected as being less exposed to injury whilst the child is being carried). Avoid drawing blood if possible. Then smear the scarification with lymph taken from the vaccinifer and allow it to dry.

Precautions—In view of the increasing objection to vaccination certain precautions are necessary. (1) If possible the arm of the child who is to be vaccinated should be cleansed at the point of inoculation. (2) The lancet, needles, or other instruments which are used should be free from rust, carefully cleansed and made aseptic. (3) Lymph should be taken only from a child who is quite free from disease or

hereditary taint. Therefore reject all lymph from a syphilitic or a strumous child, since it has been demonstrated with disastrous results that syphilis, erysipelas, and other blood diseases can be communicated by means of vaccination. (4) Lymph should only be taken for inoculative purposes from children who are the subjects of primary vaccination. (5) If possible yield to the wishes of parents who desire their children to be vaccinated 'direct from the calf,' as although there is no necessary proof that the animal is in a quite healthy state and free from defective hygienic surroundings, the procedure removes one source of objection. (6) Repeat the operation at puberty, or even a third time if there be an epidemic of small-pox. The rule probably holds good that if, even after successful but remote vaccination, subsequent inoculation of the vaccine virus produces the usual phenomena of the disease, such person was not entirely immune from small-pox. (7) Inoculate the virus at the first vaccination at three and, if possible, four separate and distinct points.

CHICKEN-POX (VARICELLA, GLASS-POX)

Definition.—A specific, infectious, eruptive fever, the eruption appearing in successive crops, and passing through the stages of pimple, vesicle, and scab.

Etiology.—(a) **PREDISPOSING CAUSES.**—*Age.*—A disease essentially of childhood. *Sex* has no influence. *Season.*—Most cases appear during the spring and summer months.

(b) **EXCITING CAUSE.**—A specific poison which is given off by the breath and skin of a person suffering from the disease.

Incubative Period.—Not positively known yet ; probably about ten to fourteen days.

Symptoms. **PRIMARY FEVER.**—Slight and perhaps unnoticed ; lasting twenty-four hours.

ERUPTION.—Appears on the second day, usually on the back and shoulders, then on the face, scalp, trunk, and extremities, and occasionally in the mouth. There is, however, no definite order in its appearance. The rash consists of rosy

papules, which become vesicles in twenty-four hours, irregular in size, and resembling in appearance the results of sprinkling the skin with boiling water. The vesicles, which are also occasionally umbilicated, then become filled with a turbid serum, and in one to two days after they dessicate. The eruption does not become pustular, except under the influence of irritation. The scab falls off three days later, leaving no scar, unless the eruption be irritated from picking or pressure.

During the whole of the eruptive period the fever is slight (101° F.), and there is some malaise and languor.

Diagnosis.—FROM SMALL-POX, by absence of severe lumbar pain and fever, and by the rash passing from vesicle to scab.

Complications.—None. Occasionally the pulmonary complications common to all febrile disease may occur.

Treatment.—Keep the patient in bed. Give a saline febrifuge, or a laxative if necessary. Prevent the patient picking the eruption, which may be smeared with cold cream or with carbolised oil.

SCARLET FEVER

Definition.—An acute, specific, eruptive disease attended by sore throat. The eruption consists of a general, diffused, and punctiform blush, followed by desquamation.

The term 'scarlatina' should not be used. It is generally applied to mild forms of scarlet fever, and is, therefore, misleading.

Etiology.—(1) PREDISPOSING CAUSES. —*Age.*—Essentially a disease of early childhood (under five years), although adults may suffer, especially if their childhood was passed without the fever. *Season.*—It prevails especially in autumn, and then often in epidemic form.

(2) EXCITING CAUSE. —A specific poison given off by the breath, skin, and excretories, or by fomites (such as bed-clothes, garments) of an infected person. It may, therefore, be inhaled or taken in by the mouth, by milk and other foods. The infectious character of the disease is very marked and lasting. Cases are recorded in which the fever has been con-

veyed to distant parts by letters and by clothes.¹ *Micrococci*. have been found in the blood and other tissues, but apparently these are not specific.

Incubative Period.—From twenty-four hours to five days. The latter duration is rare. In our own case the first symptom of fever commenced on the third day after exposure.

Symptoms.—PRIMARY FEVER.—Begins with chills, rigors, lassitude, vomiting, and, occasionally, convulsions (in children).

ERUPTION appears at the end of the first day. It consists of a general scarlet blush, disappearing on pressure, interspersed with minute asperities corresponding to hair follicles. The rash, which often causes great itching, may be compared to the appearance of red blotting-paper as seen through an ordinary lens. It first appears generally at the pit of the stomach, in the armpits, or at the flexor surfaces of the elbows, where the skin is soft and delicate. It lasts usually four days, when it gradually fades and disappears, and is gone by the seventh or eighth day. This is followed by desquamation, which is not entirely completed till the end of the fifth or even sixth week. Desquamation occurs in large flakes, especially from the palms and soles; indeed, a complete cast of the hand may be shed. The dead cuticle, however, lingers between the toes and fingers, especially on their dorsal aspects. The eruption also attacks the throat and fauces. There is injection and swelling of the tonsils, with a sense of soreness and fulness, the voice consequently being thick and 'leathery.' The sore throat, however, is mainly pharyngeal.

OTHER SYMPTOMS.—The temperature rises on the first day, reaching 100° F., or higher. On the second day, with the appearance of the rash, it is still higher (103° to 105°), and continues much at the same level till the sixth day, when it suddenly falls to about 99°, or even to normal, unless there be some complication. The fever thus terminates by crisis.

¹ Murchison records a striking example. A woman in Scotland attended her child, who was ill with the fever. The illness was fatal. Immediately afterwards the mother departed for India, taking some of her child's clothing in a box. The box being opened in the Red Sea caused a severe epidemic of scarlet fever on board ship.

The tongue is characteristic. At the end of the primary fever it is covered with thick white fur, through which the injected papillæ protrude and stand out as red points (strawberry tongue). Three days later the tongue clears, and continues to do so rapidly, till it is abnormally red, with the projecting papillæ still apparent (raspberry tongue). This latter condition is almost pathognomonic, and should lead us to suspect scarlet fever if the disease be epidemic, even though no other sign be present.

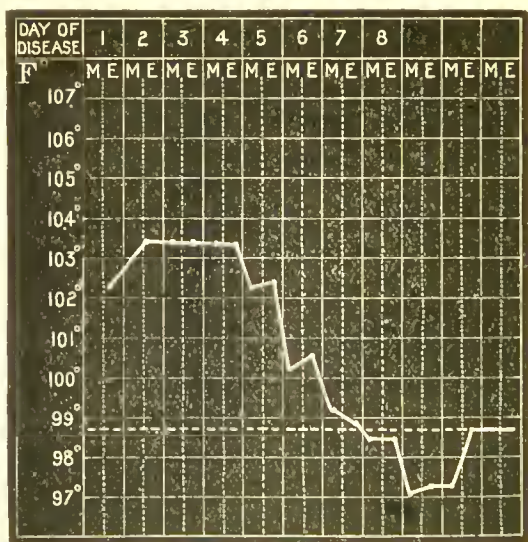


FIG. 2.—TEMPERATURE IN SCARLET FEVER.

The pulse rises in frequency with the temperature, reaching 120 or more per minute. Respiration is hurried; the patient is dull and apathetic, whilst delirium is often present, especially at night. The urine is concentrated and scanty, with diminished chlorides and increased urea.

Diagnosis.—FROM MEASLES. By the absence of coryza and catarrh of the respiratory tract before eruption. The rash, too, is uniform and appears early, whilst in measles it consists of dusky pink papules, and is not apparent till the

third or fourth day. The sore throat of measles is laryngeal ; that of scarlet fever is pharyngeal.

FROM DIPHTHERIA.—In this disease albuminuria is always an early symptom (Murchison) ; there is no eruption, and, as a rule, the throat symptoms are marked by a characteristic exudative membrane.

FROM ERYTHEMA, which occasionally attends septicæmia and the administration of certain drugs (copaiba, iodides, bromides, belladonna, &c.). In all these conditions, except septicæmia, a marked rise of temperature is rare, and the diagnosis is easily settled by the history of the case.

FROM TONSILLITIS.—This disease is attended by marked inflammatory swelling of one or both tonsils, accompanied by a sticky exudation, and there is no rash.

Complications.—1. ACUTE TUBAL NEPHRITIS, which may supervene early, but is not usually found till the end of the fever. Arterial tension, with accentuation of the aortic second sound, together with scanty secretion of smoky urine, should always lead to examination for albuminuria.

2. RHEUMATISM.—Often present from the beginning, and then a distressing symptom. It may be followed by pericarditis and endocarditis, with permanent lesion of the heart valves. Indeed, as a cause of valvular disease scarlet fever is more important than is generally credited.¹

3. THROAT AND CELLULAR CUTANEOUS SYSTEM.—Sloughing of glands of neck, of tonsils ; necrosis of mucous membrane and perforation of cheek (*noma parotidis*) or of vulva (*noma vulvæ*). The two latter are, however, rarer than in measles.

4. EAR.—Inflammation of tympanum, which may lead to necrosis of ossicles, or suppuration with perforation of membrana or of roof of tympanum.

5. RESPIRATORY TRACT.—Edema of glottis, pneumonia (both forms), bronchitis, pleurisy (with or without effusion). The latter occurs especially towards the end of the fever.

6. PARTURITION.—Puerperal scarlet fever, a most grave

¹ Note, also, the frequent association between throat affections (scarlet fever, quinsy, hospital sore throat) and inflammation of joints.

complication, constituting one of the forms of puerperal fever, and is generally fatal.

7. INFLAMMATION OF SEROUS CAVITIES, with serous effusion into pleuræ, pericardium, or peritoneum.

Varieties.—(i) *Sc. F. Mitior*.—In which the symptoms are mild and may be overlooked. The signs may be confined to a slight throat affection, but with no rash; or, on the other hand, the rash may be well marked and the throat phenomena almost absent.

(ii) *Sc. F. Anginosa*.—In this there is high fever (106° to 107° F.), the throat symptoms are very pronounced, the parotid and submaxillary glands involved, with brawny swelling of the neck; vomiting and diarrhœa are often urgent. The rash is occasionally deferred till the third day and is patchy and darker in character. Head symptoms, such as high delirium followed by stupor and coma, are usual.

(iii) *Sc. F. Maligna*.—Characterised by the absence of rash or its rapid disappearance (vulgarly 'gone inwards'); or its conversion into petechiæ in some instances; by fever of a low typhoid type (with dry tongue, muttering delirium, diarrhœa); by sloughing of tonsils; an ærid, fœtid discharge from the nostrils, and otorrhœa; by swelling of glands of neck (always) and deep-seated abscesses.

It may be remarked that at times there is a difficulty in classifying a given case under any of the above headings. It may present features which would place it in any of these categories. Nor should it be forgotten that a mild case may communicate the disease to another in a most malignant form, the severity of the disease apparently depending mainly on the constitutional state of the recipient.

Prognosis.—Always a grave disease, since, however mild the attack, it may be followed by fatal sequelæ. The death-rate from scarlet fever has taken the place formerly occupied by small-pox.

Treatment.—Remove all curtains, carpets, and superfluous furniture. All the bedding, clothes and personal linen should be boiled, or otherwise disinfected.

The treatment of a mild attack demands little more than

saline febrifuges, an occasional laxative, together with antiseptic gargles and washes ¹ for the mouth. Our chief precautions should be directed (1) to obviate complications and sequelæ; (2) to prevent infection spreading to other inmates of the house. Murchison recommended that no patient be allowed out of bed under a month, and not out of his room under six weeks. The chest should be daily explored with a view to detect cardiac or pulmonary affections, and the urine should be examined daily for albumin. In view of the eminently infectious character of the disease, especially during desquamation, it has been recommended that the body should be well anointed with carbolised oil (1 in 25). We have, however, found better and speedier results by the use on alternate days of a hot bath to which a handful of ordinary washing soda has been added. This causes speedy desquamation, but the precaution should be taken of ordering the patient to sleep between blankets or in a flannel gown. Any greasy application to the skin tends to check diaphoresis, and for this reason we do not recommend inunctions. The room should be well ventilated, and maintained at a temperature of about 60°.

In the high fever of *Sc. F. Anginosa*, we may resort to a graduated bath (100° lowered to 70° in twenty minutes), or to the wet pack at a temperature of about 65°, or to tepid sponging. We cannot, however, advise any such procedures in the presence of bronchial or pulmonary symptoms.

In this variety free stimulation is generally required (alcohol, carbonate of ammonia), together with plenty of nourishing food (milk, eggs, strong beef extracts).

Severe throat affections may be treated by swabbing the parts freely with a solution of nitrate of silver (gr. v. ad ℥j.); or a spray of antiseptic lotion (sulphurous acid 1 in 500, carbolic acid 1 in 50) should be applied freely to the nares and throat. If the patient can use a gargle, one containing free chlorine or potass. permangan. (gr. ʒ ad ℥j) is excellent. Implication of

¹ R. Potass. Chlor. ℥j.; Acid: Hydrochloric: fort: mviij.; Aquam ad ℥viij. Misce, fiat Gargar. Pour the acid on the potash salt, and then add the water, which is thus impregnated with chlorine.

glands of the neck and brawny swelling of subcutaneous tissues are relieved by free fomentation and poulticing. It may be necessary to evacuate deep-seated pus.

In *Sc. F. Maligna* the chief indications for treatment are stimulants and food. Give nutrient enemata if necessary. During convalescence iron tonics are especially indicated. The food should still be plain and simple. It is better to avoid butcher's meat for quite a month after the fever. Affections of kidneys and lungs, and other sequelæ, must be treated on the general rules laid down under the various diseases of these organs.

Subsequent disinfection of the patient's room, clothes, books, and utensils is absolutely imperative, as the disease will lurk even in a cobweb. Burning sulphur is, perhaps, the best way to disinfect the room.

MEASLES (*RUBEOLA*, *MORBILLI*)

Definition.—An acute, specific fever characterised by an eruption consisting of dusky, pink papules, often in crescentic shaped groups, and by catarrh of the respiratory tract.

Etiology.—(*a*) **PREDISPOSING CAUSES.**—*Age.*—A disease of childhood, although adults may suffer unless rendered immune by a previous attack. Second and third seizures, however, are tolerably frequent. *Season.*—It prevails at all times of the year, but especially in winter and spring, when it may assume an epidemic character.

(*b*) **EXCITING CAUSE.**—A specific poison, most active in the early stages, given off by the breath and mucous membranes of the respiratory tract. *Micrococci* have been found in the respiratory tract, which are not apparently specific.

Incubative Period.—Fourteen days.¹

Symptoms.—**PRIMARY FEVER.**—Lasts three days. It is

¹ This we abundantly proved by the following record. A young gentleman attended a juvenile party. He complained there of febrile symptoms which, on the next day, were seen to be those of measles. Ten or twelve children in different families who were guests at the party sickened with febrile symptoms fourteen days afterwards, and all developed typical measles.

characterised principally by catarrh of the respiratory tract and its offshoots. Thus we find sneezing, coryza, conjunctivitis, swollen eyelids, photophobia, hoarseness, and cough. In addition, the skin is dry, the tongue furred, the pulse is quickened, the urine scanty, the temperature raised (100° to 102°), and there are rigors, with other symptoms common to a specific febrility. Delirium, convulsions, and diarrhœa are also frequent concomitant symptoms, especially in children.

STAGE OF ERUPTION.—Arrives on the fourth day. The rash consists of papules slightly raised above the skin, at first

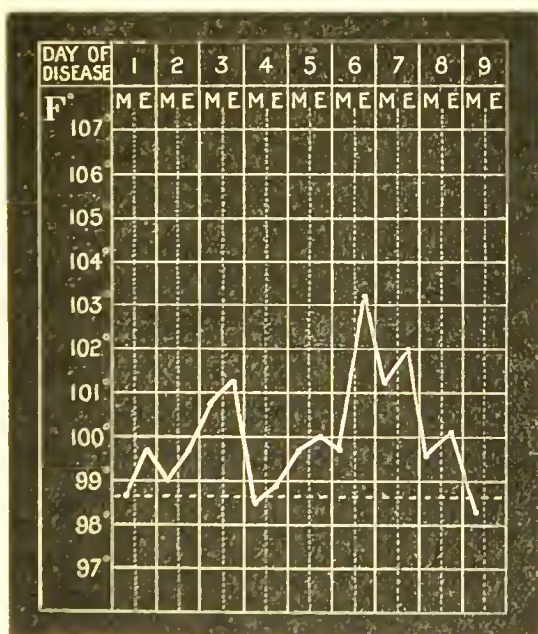


FIG. 3. TEMPERATURE IN MEASLES

somewhat resembling flea-bites owing to subcutaneous engorgement of the capillaries. As the papules increase in size they tend to coalesce, or to be arranged in closely aggregated groups. The eruption usually first shows itself on the forehead, and then in three days extends to the chest, trunk, and extremities in the order named. At the same time the soft palate, and buccal cavities generally, are red, injected, and distinctly maculated, and there is a sense of discomfort about the throat. The sore

throat, however, is mainly laryngeal. The febrile symptoms also increase with the appearance and development of the rash, the temperature, which had fallen about the third day, rising to 104° on the seventh or eighth day (third or fourth of eruption), when a period of crisis occurs, unless there be complications. Then the temperature suddenly falls to 100° , the eruption begins to fade in the order of its outbreak, disappearing by the tenth day, and is followed by a fine 'branny' desquamation. Diarrhœa, if it has not already supervened during the primary fever, frequently accentuates the crisis.

STAGE OF DESQUAMATION AND CONVALESCENCE dates from the fall of the temperature. Desquamation is not a marked feature, as in scarlet fever. Still, it may always be observed after much eruption. It is complete, probably, at the end of the second week (twenty-first of illness).

Varieties.—(1) **SIMPLE MEASLES**, as above. (2) **MALIGNANT MEASLES**, in which the prostration is intense, the eruption dark and petechial from the first, or suppressed entirely ('gone inwards'). The fever is of a low, typhoid character, with delirium. Complications supervene early, especially broncho-pneumonia and diarrhœa. Certain epidemics are characterised by this type.

Pathology.—No very distinctive lesions. There is a general congestion of the trachea, bronchi, and smaller air passages; also a catarrhal affection of the mucous surface of ileum and colon. The spleen is often enlarged and friable, and the blood dark, with a tendency to remain fluid. Beyond the above, we should only expect to find the lesion, such as broncho-pneumonia, which was the actual cause of death.

Prognosis.—Favourable as a rule. It will, however, be much influenced by the severity and type of the attack, the constitution of the patient, the previous state of his health, and the presence or absence of complications. The disease is very fatal in poor, ill-nourished children, and in Negro races.

Diagnosis.—From *Typhus* (see p. 135); from *Rötheln* (see p. 116); from *Scarlet Fever* (see p. 110); from *Tonsillitis* (see p. 210).

Complications and Sequelæ.—(1) **RESPIRATORY TRACT.**—Most frequent: viz. laryngitis, bronchitis, broncho-pneumonia,

and pleurisy. The fever also frequently renders tubercular phthisis active in those hereditarily predisposed. Whooping cough is also a frequent sequel when there has been exposure to this disease.

(2) SKIN AND SUBCUTANEOUS TISSUES.—Noma ; especially attacking the mucous membrane of the cheek (*n. parotidis*), the vulva, or the skin on front of the chest. As a rule, it only occurs in delicate, unhealthy children. Abscesses may form in different parts of the body.

(3) ALIMENTARY TRACT.—Urgent vomiting or diarrhoea.

(4) SPECIAL ORGANS.—*Eye*.—Strumous conjunctivitis. *Ear*.—Chronic inflammation of the middle ear (rare).

Treatment.—Nothing is required in an ordinary case beyond rest in bed and maintenance of a warm temperature (60°) of the room, from which also exclude any glaring light. The diet should be restricted to milk, eggs, and meat essences. The cough and catarrhal symptoms generally will be relieved by hyoscyamus, with oxymel of squills, or ipecacuanha, or tolu.¹ Gargles and diluent drinks may be allowed freely. The bowels should act regularly, but purgatives should be given with care. For the treatment of complications which may arise, refer to the chapters on the various diseases.

During convalescence give iron tonics, and in those cases with hereditary tendency to tubercle, it may be necessary to recommend a course of cod liver oil, or a prolonged sea-voyage.

GERMAN MEASLES (RÖTHELN, EPIDEMIC ROSEOLA)

Definition.—A specific, infectious fever, occurring in epidemics, with an eruption like that of measles, but with other symptoms resembling scarlet fever.

By some it has been considered a hybrid between measles and scarlet fever ; but the disease reproduces itself only.

Incubative Period.—Uncertain ; varying, according to different authors, from five to twenty days. Bristowe fixes it at five days.

¹ ℞. Spt. Ether : Nit. mʒ. ; Oxymel : Scillæ mxx. ; Mucil. Acaciæ mxx. ; Aquam ad ʒij. Misco.

Etiology.—**EXCITING CAUSE.**—A specific poison given off by the breath and skin.

Symptoms.—**PRIMARY FEVER.**—Lasts two to three days, resembles somewhat the similar stage in measles. Thus, there are rigors, running from the eyes, cough, sneezing, a dry rather than a sore throat, and at times diarrhœa or convulsions. As a rule, however, the symptoms and the fever are milder than in measles, the temperature not reaching above 101° F.

ERUPTIVE STAGE.—Characterised by the appearance, on the second day at latest, of dusky, red papules, only slightly raised above the skin on the face, arms, and chest; thence they extend to the rest of the trunk and legs. Hoarseness, cough, and laryngeal symptoms are increased with the outbreak of the rash, although the temperature falls, frequently coming down to normal. The rash lasts five days, then gradually fades, and is followed by a slight desquamation. There is generally also some enlargement of the cervical glands.

Complications.—None important. Lung affections may supervene, but not as a rule.

Diagnosis.—**FROM MEASLES.**—By the fever being shorter; coryza is not so marked; the temperature falls on the appearance of the rash. An attack does not give immunity against true measles, and *vice versa*.

FROM TONSILLITIS.—This disease is not accompanied by any eruption.

Treatment.—No treatment is required beyond rest in bed in a warm room. Saline expectorants and gargles may be given to relieve the throat symptoms, as in measles.

MUMPS (CYNANCHE PAROTIDEA)

Definition.—An acute, specific, infectious disease, occurring mostly in epidemics, and characterised by inflammation of the parotid and salivary glands, ending in resolution.

Etiology.—(a) **PREDISPOSING CAUSES.**—*Age.*—It occurs mostly in children and young adolescents. *Season* appears to exert no influence.

(b) **EXCITING CAUSE.**—A specific poison, apparently exhaled by the breath.

Incubative Period.—Varies from eight to twenty-one days.

Symptoms.—The onset of fever is sudden, and is marked by general malaise, rigors, vomiting, and increased temperature (100° to 103° or 104°). This is followed in from two to three days (it may be less) by enlargement of the parotid and submaxillary glands. The tumefaction is red and painful, causing difficulty in opening the mouth, and therefore pain in mastication or in yawning, whilst the salivary secretion is arrested at first and increased afterwards. The inflammation of the salivary glands is not necessarily symmetrical; it may attack one side and then subside, to be followed by a similar affection of the opposite gland. In the case of the parotid, at least, the main body is first involved, causing obliteration of the groove behind the lower jaw; thence it extends to the socia parotidis and to the neighbouring lymph glands. Desquamation over the surface of the glands sometimes occurs. The fever which marked the onset of the disease subsides as the salivary glands become involved.

Pathology.—The salivary glands are inflamed, causing proliferation of the epithelium of the alveoli, and cessation of secretion. The inflammation rarely goes on to suppuration.

Complications.—1. **METASTASES.**—One of its characteristic features. Inflammation may occur in the testes (orchitis), in the ovaries (ovaritis), or in the mammae (mastitis). Or, after one of these glands has become affected, the disease may even recur in the parotid. 2. **RHEUMATISM**, with sequence of endo- and pericardial inflammation. 3. Occasionally **ALBUMINURIA**.

Prognosis.—Favourable. Permanent injury, however, may occur to testes or ovaries.

Treatment.—**GENERAL.**—Rest in bed; order a slight laxative if the bowels be confined. Relieve sleeplessness by bromides or by hydrate of chloral.

LOCAL.—Apply hot fomentations, with or without opium, to the affected regions. Liniments are not advisable. If suppuration should occur the pus should be evacuated early.

Iron and cod-liver oil are often indicated during convalescence.

WHOOPIING COUGH (PERTUSSIS)

Definition.—A specific, infectious disease mostly in children, characterised by cough, occurring in paroxysms, consisting of frequent, short, and forcible expiratory efforts, followed by a long inspiration which produces the diagnostic ‘whoop’ or ‘crow.’

Incubative Period.—Unknown. According to Henoch it lasts from ten to twelve days.

Etiology.—1. **PREDISPOSING CAUSES.**—*Age.*—Early childhood, especially between the second and the sixth year. It rarely affects adults; yet mothers and nurses occasionally contract the disease in a mild form. *Season.*—More prevalent in winter and spring; and often in epidemics. It is frequently associated with *Measles* (West).

2. **EXCITING CAUSE.**—A specific poison given off by the breath, and infecting the respiratory tract. A paroxysm may be excited by irritation of the fauces or windpipe, or by distension of the stomach. There is also a distinctly neurotic element: seizures being induced by their occurrence in others, and by fear or anger.

A second attack is rare.

Symptoms.—Three stages of the disease. 1. **CATARRHAL STAGE.**—In this stage there are no symptoms which differentiate it from ordinary tracheal or bronchial catarrh. There is some wheezing, and dyspnoea, with cough, which often ends with retching. Such signs, however, would be suspicious if occurring in a child during an epidemic of whooping cough. This stage lasts from ten to twelve days.

2. **CONVULSIVE STAGE.**—Characterised by diagnostic attacks of cough. The paroxysms are frequently preceded by a form of aura, such as restlessness, beating of arms, or even vomiting. Then ensues the bout of coughing, ending in the ‘whoop’ or ‘crow,’ due to spasm of the glottis. The attack is not necessarily terminated at the ‘whoop.’ It may begin again, or there may be three in sequence. During the paroxysms the face gets red, there is general cyanosis, increased action of the

respiratory muscles, and lachrymation. It is followed by exhaustion, more or less marked. In the twenty-four hours the seizures may vary in number from ten to thirty, or even more. They are, as a rule, more frequent at night. It is important to note their frequency as a guide to prognosis and treatment. The duration of this stage is about four weeks.

3. STAGE OF DECLINE.—The cough continues and is still paroxysmal, and is attended by redness or bloated aspect of the face; but the ‘whoop’ is absent, or only rarely heard. This stage lasts from two to three weeks, and leaves some general bronchial catarrh, which has been present throughout, as a sequel.

Diagnosis.—The onset of the disease may be difficult to detect. If there is a history of exposure to infection, and if there is a severe cough in paroxysms, worse at night and attended by vomiting, we must look on the case with suspicion, and order isolation.

When the cough is well developed it may be confounded with *Laryngismus Stridulus*, but in the latter disease, although there is a series of expirations, each one is attended or followed by an inspiratory ‘crow.’ Whilst in *Pertussis* there is a long sequence of expiratory coughs, to which succeeds one loud and characteristic inspiratory ‘whoop.’

Prognosis.—Extremely fatal in infants under one year. Mortality less marked in children who are older and stronger. Complications affecting the lungs are the chief causes of danger.

Complications and Sequelæ.—1. THOSE MAINLY DUE TO THE VIOLENCE OF THE EXPIRATORY EFFORT, viz. Interlobular emphysema; hæmorrhages from the lungs, conjunctivæ, ears, gums; vomiting; diarrhœa, prolapsus ani, herniæ; ulcer of frænum lingvæ, from friction against the lower incisor teeth; incontinence of urine.

2. RESPIRATORY TRACT.—Broncho-pneumonia, pleurisy, phthisis, and caseation of bronchial glands. Measles must also be included under this heading.

3. CARDIAC SYNCOPE. From exhaustion of heart muscle.

4. NERVOUS SYSTEM.—Convulsions.

Treatment.—There appears to be no specific remedy. Bromides, belladonna, sulphate of zinc, chloral, oxalate of cerium, conium, have all been advocated, with a view to arrest seizures. Morphia ($\frac{1}{60}$ grain) appears to give the best results (Henoch).¹ It must, however, be carefully watched. In view of the germ theory of the disease, children have been ordered to reside near gas-works, or to play near the refuse heaps from the gas retorts, the vapours from the ammoniacal compounds being supposed to be antidotal. Carbolic spray and vapour of eucalyptus have been prescribed for similar reasons ; but pure air is probably more beneficial.

Attention to diet is an essential. It will be found that patients are better with a restricted quantity of food, given in small meals, at comparatively short intervals. Milk, eggs, and farinaceous puddings are best for children ; butcher's meat being avoided.

TYPHOID FEVER (ENTERIC FEVER, ABDOMINAL TYPHUS)

Definition.—An acute, specific, eruptive fever lasting over fourteen days, characterised by an eruption of rose-coloured spots, vanishing on pressure and appearing in successive crops ; and by diarrhœa, hæmorrhage, distension of the belly, and other signs pointing to the small intestine as the seat of the principal lesion.

Etiology.—(a) PREDISPOSING CAUSES.—*Age.*—Commonest in early adolescence, between fifteen and twenty-five years ; rare after fifty (this is supposed to be due to atrophy of solitary and agminate glands). Nevertheless all ages may suffer. *Climate and Season.*—Most prevalent in warm and temperate regions. In Britain it occurs chiefly in late summer and autumn, when the rains have washed the surface drainages and decompositions into wells. This is often spoken of as the 'autumn epidemic,' which begins at the end of July, increases in severity with each succeeding month till November, when it declines as the cold weather sets in. In spring, therefore,

¹ R. Liq: Morphine Acetatis ℥ij.; Oxy-mel: Scillæ mxx.; Aquam ad ʒij. Misc.

the disease is rare, although, to a certain extent, it is endemic all the year round. *Locality* exerts no influence. It prevails in towns as well as in the country. Its prevalence of late years has by some been ascribed, in a measure, to the existence of intramural water-closets. *Station of Life*.—All ranks, from prince to peasant, are liable to the disease. *Constitutional Defects* do not appear to exert much influence; on the other hand, the disease appears, in certain epidemics, to attack the robust and the 'newly arrived' to an infected district.

Second attacks may occur.

(b) **EXCITING CAUSE**.—A specific poison given off by the excreta of a typhoid patient and entering the alimentary canal of its victim. Koch has described a bacillus of typhoid which is present in the ulcers, also in the mesenteric glands, spleen and other organs. The bacilli occur in groups, each individual being rod-shaped, two to three μ in length, and a third as broad, with distinctly rounded off ends. They bear spores, either occupying the whole breadth of the organism, or at the ends only.¹

Wells, streams, and other sources of drinking water, also milk, may be contaminated by typhoid sewage, the bowel thus becoming a continuation, as it were, of the sewer. It has also been abundantly proved that sewer gas itself may give rise to the fever. Two cases of direct infection by inhalation of the effluvia of typhoid evacuations have been observed by us. But it is probable, both in the case of sewage and sewer gas, that such contamination is due entirely to the presence of typhoid evacuations, or to decomposing feculent matter.

Incubative Period.—Uncertain; probably about twelve days as an average.

Symptoms.—**DURING THE FIRST WEEK** the symptoms are mild, constituting a kind of primary fever. The patient suffers from malaise, he feels chilly and wishes to sit near the fire; he has headache, the bowels are irregular, this often being exaggerated by some aperient medicine which the patient has taken. In many cases it will be found that he has followed his usual daily occupation, although stricken with fever for the past four or five days.

¹ See *Micro-organisms*, by Flügge. New Sydenham Society.

As the disease advances he becomes restless at nights, when the fever is increased, the skin being hot and dry, though moister in the daytime. The pulse has risen in frequency to

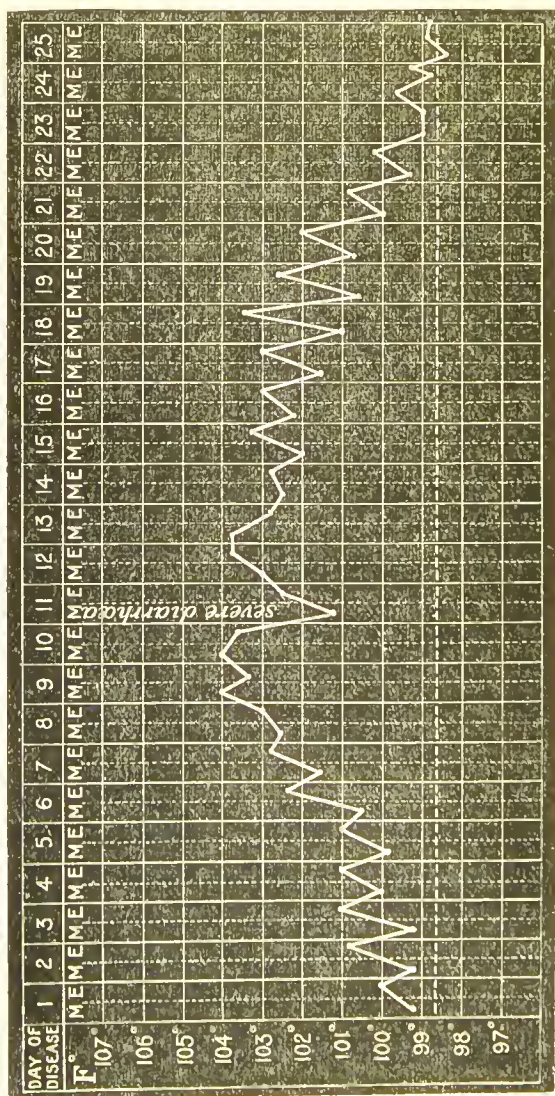


FIG. 4.—TEMPERATURE IN TYPHOID FEVER

100 or 120. The temperature also has gradually increased, showing its intermittent character by rising two degrees in the evening and falling one degree in the morning. At the end of the first week the evening and morning observations range from 102° or 104° , to 100° or 101° respectively. The bowels are loose, the evacuations being alkaline and offensive, consisting mostly of bile.

IN THE SECOND WEEK the fever advances. The lips are parched, the tongue is dry, furred on the dorsum, but clean at the tip and edges. The face is flushed; there is pain, tenderness, and gurgling in the right iliac fossa; the spleen is enlarged, and the belly distended. Epistaxis is also a common symptom. The mind keeps clear. The characteristic eruption makes its appearance between the ninth and twelfth days. It consists of rose-coloured, elevated spots, disappearing on pressure, scattered over the abdomen, chest, and back, and probably found on the limbs if carefully searched for. As a rule, however, the rash is scanty, and often not found or overlooked. The spots appear in successive crops, each lasting about four days. The stools are still alkaline and loose, but have now assumed the typical pea-soup colour and consistency.

IN THE THIRD WEEK the temperature has reached the summit of the curve, with a daily variation of two degrees (104° P.M. to 102° A.M.). Sometimes this is reversed, the morning reading being higher. The patient is drowsy and deaf, he is delirious at night, the pulse is feeble, the tongue dry and fissured, the pupils are dilated, diarrhoea and other abdominal symptoms continue. Towards the end of the third week the strength has much diminished, there are tremors, subsultus, muttering delirium, a dicrotous pulse, and all the symptoms which constitute the so-called 'typhoid state.' He is, indeed, now suffering from the effects of a septicaemia. At this period obstinate vomiting, or diarrhoea, or perforation may take place. The rash is still apparent.

IN THE FOURTH WEEK, unless complicated by a relapse, there is an amelioration of all the symptoms. The patient sleeps better, the heavy stupor passes off, the face is pale and shrunken, the tongue is moister, and the stools darker and of

thicker consisteney. The temperature also falls, the intermissions being less marked, until finally, about the twenty-sixth or twenty-eighth day, it is normal in the morning, although the evening record may still be high (101° or more). Convalescence cannot, however, be said to have set in until the morning and evening temperatures are both normal on two successive days.

During the whole of the fever there are, in addition, all the symptoms common to every specific fever. The urine is scanty, of high sp. gr., and contains an excess of urea with diminished chlorides. There is some bronchial catarrh, with congestion at the bases of the lungs.

The above description will apply to an ordinary case of enteric fever; but it is a common observation, made by physicians who have studied the disease in Germany, in Egypt, or in India, that the type of enteric fever in these countries is much more severe in all its symptoms and complications than in Britain. It should also be remembered that in certain warm climates typhoid may be complicated by malarial influences which considerably modify, if they do not entirely alter, the character of the fever.

Pathology.—The characteristic lesions occur chiefly in the glands of the *small intestine* (Peyer's and solitary), and, therefore, are most apparent in the ileum, cæcum, and the upper part of the large intestine. The infection appears to start in the lower part of the ileum, and, although it extends downwards through the ilio-cæcal valve to the colon, it is more intense as it proceeds upwards towards the jejunum. The glands, infiltrated with the specific poison, become enlarged and bile-stained. Between the fourteenth and twenty-first day ulceration occurs, the ulcers being oval or circular, with thickened, irregular, and overhanging edges, and varying in size from a hemp-seed to a penny-piece, according as the solitary or agminated glands are involved. In the latter case the ulcers are situated opposite the attachment of the mesentery, and have the long axes parallel to the length of the gut; but by no means invariably: a typhoid ulcer is frequently transverse to the bowel. The floor of the ulcer is lined with

flocculent matter, composed of sloughing submucous or muscular coat, according to the depth of the excavation. If the necrosis proceed to the peritoneal coat, a local peritonitis may occur, agglutinating the part to a neighbouring coil; or the serous investment may be perforated by a pinhole aperture, causing general peritonitis and death. The perforation usually occurs about eighteen inches from the valve, or, more rarely, in the cæcum or appendix. If recovery take place, the ulcers heal in the order of their appearance, each ulcer taking about two weeks to heal, and the whole process of healing occupying about four weeks. It leaves no puckering or narrowing of gut.

The *Mesenteric Glands* are also swollen and congested.

The *Spleen* is enlarged, congested, and friable.

The *Voluntary Muscles* will be found to have undergone granular and waxy degeneration, these changes being most marked in the adductors of the thigh, and the recti abdominis. The lungs, liver, and kidneys are more or less congested.

Complications.—(a) GASTRO-INTESTINAL. — 1. *Relapse*, being a return of the fever (re-infection), with spots and other typical symptoms. The attack may be severer than the initial fever. A second relapse is not uncommon. 2. *Recrudescence*, a form of abortive relapse, characterised by a slight return of pyrexia and, possibly, diarrhœa. 3. Severe *intestinal hæmorrhage*, indicated by a sudden fall of temperature and the appearance—it may be at once, or the next day or two—of mælena. 4. Urgent *diarrhœa*, also causing a marked fall of temperature. 5. Constant *vomiting*, which should make one suspect peritonitis. 6. *Perforation* and *peritonitis*, shown by abrupt fall of temperature, thoracic respiration, drawing up of thighs on the abdomen, a feeble, wiry pulse, and a drawn, anxious expression. 7. Excessive distension of the intestines with gas (*Meteorism*). 8. *Jaundice* (rare).

(b) RESPIRATORY TRACT. Ulcer of the larynx (generally in the neighbourhood of the arytenoid cartilages), bronchitis, broncho-pneumonia, pleurisy, activity of latent tubercle.

(c) CIRCULATORY SYSTEM. Profuse epistaxis, embolism, and thrombosis of veins, which causes œdema.

(d) CUTANEOUS AND CONNECTIVE TISSUES.—Sloughing of

skin over sacrum or scapulæ, abscesses, glandular enlargements, alopecia.

(*e*) OSSEOUS SYSTEM.—Periostitis, nodes, necrosis.

(*f*) NERVOUS SYSTEM. — Deafness, convulsions, wild delirium, post-febrile dementia, melancholia, delusions, catalepsy.

(*g*) URINARY SYSTEM.—Cystitis, albuminuria.

(*h*) HYPERPYREXIA. —Always a grave symptom. From records of many cases, we find that a patient has seldom recovered who has presented at any time during the course of the fever a temperature of 105° or upwards.

Finally, we conclude that no fever taxes a patient's strength so much as typhoid does. It seems to detect any weak spot in his constitution, and to render active any latent disease or constitutional taint.

Diagnosis.—FROM TYPHUS.—See table, p. 135.

FROM PNEUMONIA.—Sometimes difficult. We must, however, depend on auscultation, the presence of rusty sputa, of labial herpes, and the absence of spots and enteric symptoms.

FROM GENERAL ACUTE TUBERCULOSIS. — Inquire into family predisposition to tubercle; examine sputa (if there be any) for 'bacillus tuberculosis'; examine the choroid for tubercle. The belly in tubercular ulceration is usually retracted, whilst the skin is dry and harsh. The diagnosis between the two diseases is often, however, a matter of extreme difficulty.

FROM TRICHINOSIS. — Here there would be puffiness of eyelids and face, muscular pains, but no spots, and no splenic enlargement.

FROM OTHER SPECIFIC FEVERS.— There are three points which clear up the diagnosis, viz. : (i) the fever having lasted longer than a week, with no symptoms of a local inflammation to account for it, and with no eruption characteristic of measles, scarlet fever, or small-pox; (ii) the type of the fever is remittent, gradually becoming intermittent; (iii) the presence of lenticular rose spots. (Murchison.)

Prognosis.—Should always be guarded, however mild the attack appears to be, since the patient's life may be destroyed by relapse of a severe type, or by grave complications and

sequelæ. The death-rate varies from ten to twenty-five per cent., and is probably higher in hospitals, owing to the enfeebled health of the patients, than in private practice amongst the well-to-do. The graver symptoms are, high fever (105°) with slight morning remissions; urgent vomiting after the fourteenth day; profuse diarrhœa; marked meteorism; severe tremors (indicating deep ulceration); a sudden fall of temperature, with signs of perforation; relapse, especially if the symptoms of the new attack be severe, as the patient is in a condition less able to withstand the fresh onset.

Treatment. — Perhaps no disease has caused so much diversity of treatment. Some practitioners advocate ‘doing nothing’ beyond giving a slight saline febrifuge; others prescribe acids (dilute hydrochloric, $\text{m} \times . \text{t. d.}$), with a view to counteract the alkalinity of the evacuations; again, others advocate gentle purgation ($\text{p. hydrarg. c. creta, gr. iij. t. d.}$), with a view to eliminate the poison, and excellent results appear to follow. It is unquestionable, however, that the best line of treatment consists in maintaining the patient’s strength and conducting him through the fever. Still, the best authorities agree that disastrous results may occur from a ‘do-nothing’ policy as from one which is meddling. The symptoms must be treated.

The room should be warmed and well ventilated, and, if possible, of good dimensions. Give food freely, in small and frequent quantities. Eggs, milk, and beef-tea should constitute the chief dietary, and should be continued right through the fever. Solid food (boiled fish or fowl) has been given three days after the temperature has fallen to normal; but we prefer to wait at least a week. Stimulants (brandy) should only be ordered when required, their exhibition being demanded if the pulse be frequent and feeble or dicrotous, if there be much delirium, or if subsultus and other signs of exhaustion be present. The quantity of brandy which can be tolerated by a patient will vary considerably. Four ounces in twenty-four hours may be sufficient, but we have seen cases which required, and in whom recovery was apparently due to, sixteen ounces. Examine the bladder daily to see that there

is no retention. Prevent bedsores by scrupulous cleanliness and change of bed-linen. Immediately remove and disinfect evacuations. Further remedial measures must be directed against those symptoms which by their exaggeration become complications.

Thus, *Diarrhoea*, if excessive (over six stools in the day), should be checked by enema of opium, dilute sulphuric acid (℥x.), and tincture of opium (℥x.), catechu, or the extract of hæmatoxylum.

For *Vomiting*.—Order turpentine stupes to the epigastrium; and give dilute hydrocyanic acid (℥iij.) with effervescent alkalies, or bismuth, or lime-water. Urgent vomiting after the second week is especially grave, often preceding (? causing) perforation.

For *Constipation*.—Diminish the quantity of milk. Give castor oil, or other laxative, or order enema of olive oil. Avoid irritant purgatives, which cause excessive peristalsis.

For *Tympanites*.—Enema of turpentine; assafœtida.

For *Hæmorrhage*.—Mineral acids; opium, turpentine, ergot.

For *Sleeplessness and Delirium*.—Opium, hydrate of chloral (if the heart-sounds be strong), bromides.

For *Perforation and Peritonitis*.—Morphia, in large doses. Of late years the question of enterectomy has been advocated.

For *Hyperpyrexia*.—Quinine, tepid sponging, ice-pack, graduated bath (100° reduced to 75° in twenty minutes). The latter procedure cannot, however, be adopted without due care and consideration. It appears to be especially contra-indicated when there is a feeble circulation, with cyanosis, or when there are marked signs of pulmonary congestion and collapse.

DURING CONVALESCENCE.—Diet is still an important factor to recovery. The food should still be 'sloppy.' Avoid bread in any form until complete recovery has taken place. Records distinctly point to many cases of relapse or of recrudescence dating from the day on which bread was given. (St. Thomas's Hospital Reports, vols. ix., x., xi.)

TYPHUS FEVER (JAIL FEVER, SHIP FEVER)

Definition.—A specific, contagious fever, lasting fourteen days, with an eruption at first resembling that of measles, but soon becoming petechial. There is a tendency to stupor, delirium, and the typhoid state.

Etiology.—(a) **PREDISPOSING CAUSES.** *Age.*—All may suffer, but it especially attacks people over thirty years old (important in selection of a nurse). *Climate.*—Cold and damp (Britain, Russia, Danube provinces). *Season.*—The fever prevails in winter, when the poor herd together for warmth; it disappears as spring returns and when houses are ventilated. *Locality.*—Found only in towns and congested districts. *Station of Life.*—Affects the destitute and the poorest of the poor. It never attacks the better classes, except medical men, clergymen, and visitors amongst the poor. *Mental Depression and Anxiety, Hardships, and Fatigue.*—It prevails, therefore, amongst those struggling for life, and also in the retreating army, or the beleaguered garrison, in greater proportion than in the victorious troops.

(b) **EXCITING CAUSES.**—A specific poison given off by the body, breath, and excreta of a patient suffering from the fever. The poison enters by the respiratory tract or by the mouth. The fever may originate *de novo* from the concentrated exhalations of human beings crowded together (hence, in former times called jail fever, slave-ship fever). The disease, therefore, often occurs in epidemics supervening on famine (potato famines, Ireland; cotton famine, Lancashire), and is secondary to destitution, and is not related to atmospheric causes. Epidemics have thus been predicted, *e.g.* after the famine in Russia (1891–92).

Incubative Period.—From a few hours, in virulent outbreaks, to fourteen days. The latter is the usual period.

Symptoms. 1. **PRIMARY FEVER, INVASION.**—Comes on suddenly, so that the patient knows the day on which he sickened. It is attended by intense frontal headache, rigors,

thirst, foul tongue, and loss of appetite. It is speedily followed by muscular weakness.

2. STAGE OF ERUPTION.—Occurs on the fourth day. The rash consists of red-brown, elevated papules, which fade on pressure at first; and then, owing to extravasation of blood from the contained capillaries, the papules become converted into petechiæ, which are permanent during the whole fever, and even remain apparent after death, as a bruise does. The rash is first seen on the abdomen, chest, back, and arms; it is occasionally, but rarely, found on the face. It does not appear in successive crops, and may be scanty or abundant.

The fever increases after the appearance of the rash. The temperature, which rose at once on the day of invasion, continues to mount, and reaches its maximum on the sixth or seventh day (104° or higher), and continues high to the end, with slight morning remissions only. The pulse is correspondingly quick. Beginning at 100, it is accelerated to 120 or more on the fourth or fifth day. In character it is full at first, then becomes feeble, or it may be intermittent, or dicrotous. The heart-sounds, especially the first, are feeble. The respirations are shallow and quick. The tongue is shrivelled, dry, and brown, or possibly black; sordes collect on the teeth and lips; the bowels are constipated as a rule, and the 'typhoid' state is a well-marked feature at the end of the first week. The aspect is heavy and 'drunken-' looking (characteristic): the face being suffused, the conjunctivæ injected ('ferrety'), and the pupils contracted.

The body exhales an offensive odour (called by different observers 'mousey,' 'cheesy,' 'damp hay,' 'poor's smell'), which is almost diagnostic. The mental condition becomes obfuscated early; at the period of eruption, delirium is constantly present, being increased at night. Tremors, subsultus, a tendency to sink down the bed, and signs of general prostration are marked features; whilst, in severe cases, picking at the bed-clothes (carphologia, floccitatio), incontinence of urine, and coma vigil (a state in which the patient lies unconscious, with staring eyeballs) show the gravity of the illness.

The urine is scanty, high-coloured, and concentrated, with an excess of urea and uric acid, and diminished chlorides. Albumin is not uncommon.

3. CRISIS.—Towards the fourteenth day of the illness we look for a marked change. If the case is to end favourably the patient's delirium ceases, intelligence seems to return, and he falls into a quiet sleep from which he awakes refreshed, 'rational, and another man' (crisis). Muscular tremors have ceased; the



FIG. 5.—TEMPERATURE IN TYPHUS FEVER

temperature has fallen, almost vertically, to 98.6° or lower; the pulse regains somewhat its normal frequency and volume; and there is a deposit of lithates in his urine. From this date convalescence is rapid. The tongue cleans, the appetite returns, and there is gradual approach to health and strength. It is common for patients after recovery from typhus to express themselves as feeling better than they ever did before. The fever seems to consume all deleterious matter which had previously accumulated in the system.

On the other hand, the fourteenth day may be critical,

inasmuch as the temperature instead of falling may ascend to 106° or 107° . Profound coma is present, the respirations become more hurried, and life is extinguished apparently by grave cerebral disturbance and failure of heart's action. The temperature may even rise to 108° or more after death.

Varieties.—1. **TYPHUS BUBONIDES.**—Characterised by marked malignancy of the fever, and induration generally of the lymph glands throughout the body.

2. **TYPHUS SIDERANS.**—In which the patient is suddenly smitten by fever of a most virulent type, and dies within a few days, or it may be hours.

Prognosis.—Always a grave disease; about one in seven cases dies. Mortality is somewhat higher in males; in patients over thirty years of age; where there is a presentiment of death; when the typhoid state is well marked; when there is severe delirium, coma vigil, or carphology; when the pulse is persistently over 120, and dicrotic, especially if the cardiac systole be almost inaudible; when the eruption is dark and copious; or when severe complications arise, *e.g.* Bright's disease, pronounced pneumonia, incontinence of urine, bed-sores, and abscesses.

Complications and Sequelæ.—1. **RESPIRATORY TRACT.**—Hypostatic congestion of the lungs is almost invariably present: this may pass on to true pneumonia, or there may be bronchitis or pleurisy.

2. **VASCULAR SYSTEM.**—Phlegmasia, embolism.

3. **GLANDULAR AND CUTANEOUS SYSTEM.**—Bedsores, abscesses, buboes, especially in groins and axillæ, gangrene of hands and feet.

4. **URINARY SYSTEM.**—Hæmaturia, Bright's disease.

5. **NERVOUS SYSTEM.**—Post-febrile mania or dementia.—Occasionally met with after all febrile states, but especially typhus.

6. **NECROSIS OF BONES.**

Pathology.—There is no marked specific lesion. The blood is black, tarry, and uncoagulated; the muscles are soft and flabby; the brain is congested; and decomposition is rapid.

Diagnosis.—1. FROM PNEUMONIA, especially affecting the apex.—By the absence of petechial spots; the presence of rusty sputa; herpes on the lips, and auscultatory evidence. Inquire also for any history of exposure to infection of typhus.

2. FROM MEASLES.—This fever usually attacks children, in whom typhus is rare. There will also be signs of coryza and catarrhal affection of the respiratory tract.

3. FROM PURPURA.—This disease is non-contagious, and has no febrile symptoms. Further, the presence of hæmorrhages from the gums, and other mucous surfaces, together with general pallor of countenance would be sufficient to exclude typhus.

4. DELIRIUM TREMENS.—By the alcoholic history and the absence of rash.

5. FROM TYPHOID.

TABLE OF DIAGNOSIS

—	Typhoid	Typhus
Age . . .	Youth, under twenty-five . . .	Adults, over thirty
Season . . .	Autumn . . .	Winter
Climate . . .	Warm and temperate . . .	Cold and damp
Locality . . .	Towns and country . . .	Towns only
Frequency . . .	Common . . .	Rare
Station of life . . .	Rich and poor alike . . .	Poor and destitute
Incubation . . .	Not definitely known . . .	Twelve to fourteen days
Infection . . .	By impure water, sewer-gas, etc.	By concentrated exhalations due to overcrowding
Onset . . .	Gradual . . .	Sudden
Prostration . . .	Not great till late stages. He often walks into the ward	Immediate. He is carried into the ward
General Symptoms	Intestinal and abdominal . . .	Cerebral
Eruption . . .	Rose spots, disappearing on pressure	Dusky spots, not disappearing on pressure
Tongue . . .	Furred; clean at tip and edges	Dry, brown, or black
Bowels . . .	Diarrhœa; stools alkaline . . .	Constipation; stools acid
Belly . . .	Distended . . .	No marked signs
Pupils . . .	Dilated . . .	Contracted
Facies . . .	Hectic or icteric . . .	Heavy, dusky, drunken
Temperature . . .	Rises gradually; markedly intermittent; falls gradually	Rises suddenly; intermissions not marked; falls suddenly
Pulse . . .	Quick and full . . .	Quick, feeble, and dicrotic
Odour . . .	Not characteristic . . .	Diagnostic; 'mousey'
Mental state . . .	Generally clear. Slight delirium in second or third week	Stupor and coma characteristic. Delirium early, of a low type
Duration . . .	Over twenty-one days . . .	Fourteen days only
Convalescence . . .	Gradual (lysis) . . .	Sudden (crisis)
Complications . . .	Perforation; hæmorrhage; peritonitis	Pneumonia; cardiac failure; gangrene; buboes
Relapse . . .	Common . . .	No relapse
Lesions . . .	Ulceration of Peyer's and solitary glands; enlargement of spleen	No intestinal ulceration; spleen not enlarged; blood black and tarry

Treatment.—(a) PROPHYLACTIC.—Prevent overcrowding ; allow plenty of fresh air, by constant ventilation, in houses or hospitals where the destitute are lodged. If possible ensure personal cleanliness, changes of linen, and good food. The nurses should have plenty of food and rest, and should be careful not to inhale the patient's breath.

(b) CURATIVE.—The patient should be placed in a large, airy room, if possible at the top of the house. The disease will not extend downstairs to the rest of the inmates if the house be kept well ventilated with fresh air. Absolute cleanliness of patient and of bed is essential ; hence, a good nurse is of first importance.

The main indications for treatment are to maintain the general strength by nutritious food, and, if necessary, by stimulants. Food must be given ; and if it cannot be taken by the mouth it must be administered by enemata, but every three or four hours only, so as to allow rest between times.

There is no specific drug remedy. Salines may be given, but ammonia and decoction of cinchona, or some preparation of ether, are usually required. In cases of severe prostration sulphuric ether may be injected subcutaneously. Stimulants are always indicated in old patients, when the tongue is dry and tremulous, or when the pulse and heart-sounds are very feeble. Give four to ten ounces (or more) of brandy *per diem*, according to signs of exhaustion. Then treat symptoms as they demand, *e.g.*—

Constipation should be relieved by calomel or castor oil.

Delirium by darkening the room, perfect quiet, ice to the shaved head, and the exhibition of bromides in large doses. Opium may be given if there be no severe cerebral signs ; or hydrate of chloral, provided the heart-sounds are not too feeble. Both drugs, however, require careful watching on these grounds.

Retention of urine may require catheterisation. The bladder should certainly be attended to daily.

Convulsions are usually fatal. We should, however, give a calomel or croton-oil purgative, or diuretics, with a view to relieving renal congestion.

Other symptoms causing trouble should be treated on general grounds. Quinine and iron tonics are necessary during convalescence.

RELAPSING FEVER (FAMINE FEVER)

Definition.—A contagious, non-eruptive disease, characterised by high fever, congestion of the liver and spleen, and pains in the back and limbs. On the seventh day the fever suddenly subsides, followed by seven days of apparent convalescence, when a relapse occurs, with renewal of all the symptoms. The relapse lasts usually about three or four days. A second relapse is not uncommon.

Causation.—(a) **PREDISPOSING.**—*Age.*—All ages suffer ; but it specially affects young adolescents between fifteen and twenty-five years of age. *Season* apparently exerts no influence. *Occupation.*—Tramps, and those having no fixed abode. *Overcrowding*, intemperance, exhaustion, depression, and similar conditions which predispose to typhus. *Famine*, however, is the chief predisposing factor. It therefore occurs in epidemics when crops have failed, and precedes typhus, the one being directly due to starvation, the other due to the consequent overcrowding.

(b) **EXCITING CAUSE.**—A specific poison conveyed by the atmosphere, and by fomites. In the blood a spirillum or spiral vibrio (*Spirochaeta Obermeieri*) has been discovered. An attack confers no immunity against a second.

Incubative Period.—Varies. Evidence tends to fix it at about nine days.

Pathology. No marked lesions beyond congestion of the liver and spleen, accompanied by pigmentation of these organs.

Symptoms. Come on suddenly, and compel the patient to rest in bed at once. The invasion is marked by rigors, headache, thirst, vomiting, and lumbar pains. The temperature rises at once. On the second day it reaches 102°, or higher ; on the third day it is as high as 104° or 106°. The pulse rate is correspondingly accelerated, amounting to 110 or 130 per minute.

In addition to these symptoms we detect hæmic murmurs over the cardiac orifices. There are general muscular pains and tenderness in the abdomen, especially in the hypochondriac regions, due to congestion of the liver and spleen. Jaundice is often present. The tongue is white and moist ; there is constipation ; the urine is concentrated. But there is an absence of stupor, delirium, and rash.

On the sixth or seventh day there is a sudden crisis. The temperature falls at once to the normal level ; the tongue becomes clean ; the appetite returns ; the pains diminish ; the tenderness over the liver and spleen vanish ; and the patient is apparently convalescent.

A relapse occurs on the fourteenth day, with a return of all the symptoms attending the initial attack, but it is of shorter duration, only lasting four days.

Prognosis.—Favourable. Deaths are few, and these are due to weakness only. Pregnant women, however, generally abort and die. Convalescence is slow, and is often retarded by diarrhœa or pneumonia.

Treatment.—No special drug is required. Order a mild purgative with salines. Give stimulants if needed. The hyperpyrexia may be treated by cold baths, provided there are no signs of lung collapse or of other condition, such as great prostration, which would cause risk.

EPIDEMIC INFLUENZA¹

Definition.—An acute, specific fever prevailing in wide-spread epidemics, characterised by general pains especially located in the muscles and fasciæ, a heavy ‘drunken’ aspect of face, and marked mental depression.

Incubation.—Short. Twenty-four hours to three days.

Causation.—(a) **PREDISPOSING CAUSES.**—*Age.*—All may suffer, but it especially affects adults and people in advanced

¹ The description of this fever is based on observations of the epidemics occurring in Britain in the winters of 1890, 1891, and 1892. The disease, however, to our mind, bore a much stronger resemblance to Dengue or Break-Bone fever, common in the East.

life. *Season*.—The fever prevails in winter, especially if cold and damp. It disappears on the advent of spring and warmer days. *Station of Life*.—The records of hospitals and of private practice show that the fever is no respecter of persons. All ranks contract the disease. It is supposed to be more prevalent amongst the 'well-to-do,' but this is really not so; the apparent increase must be attributed to the greater publicity of cases affecting the 'classes.' *Occupation*.—Overwork, mental strain, and anxiety are powerful factors. The epidemic prevails amongst the toiling poor, medical men, students, overworked servants. Hence the outbreaks in hospitals have commenced with the doctors and other officials.

(b) EXCITING CAUSE.—A specific poison conveyed from one person to another by the atmosphere, or by fomites. Pfeiffer has discovered a distinctive bacillus. The disease is certainly contagious and infectious. Second or even third attacks are not uncommon. The disease appears to predispose to subsequent attacks rather than to confer immunity.

Symptoms.—The disease is marked by continued fever from the onset to termination. It begins suddenly, the patient being able to fix the day (sometimes the hour) with precision. The initial stage is marked by chills, rigors, thirst, vomiting, and loss of appetite, signs common to all febrile states. Within a few hours characteristic pains supervene. The eyeballs are sore and tender; there are aches and pains in the thighs, especially in the adductor group of muscles, and over a circumscribed area on the front of each thigh about six inches from the patella. The pains in the back extend from lumbar region to occiput, the patient feeling as though he had been beaten by a heavy stick. The temperature rises at once, reaching 103° to 104° or even higher on the second day; the face is flushed, the conjunctiva injected, and there is a heavy drunken aspect of face. A morbilliform rash is frequently seen on the trunk and limbs, lasting about forty-eight hours. The tongue is furred and flabby, the bowels constipated, and the urine scanty, with an increase of lithates. The spleen and liver are usually enlarged and tender. In addition to these symptoms there is great prostration and mental depression,

amounting at times to a fear of impending death, or to carelessness as to recovery.

After the third or fourth day the temperature falls, and rapidly regains its normal level ; the pains diminish, the appetite returns, and the patient is practically convalescent : but great muscular weakness and a cough, without physical signs, will often remain for days or even weeks after the fever has disappeared. Beyond a description of a typical case, it must be pointed out that the epidemic is marked by a large number

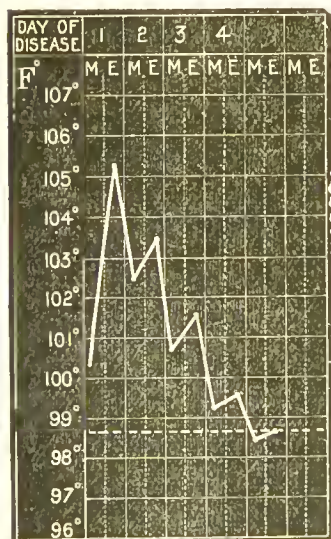


FIG. 6. —TEMPERATURE IN
EPIDEMIC INFLUENZA

of cases which, though infectious, are difficult to recognise, owing to absence of marked symptoms.

Complications are probably the most dangerous feature of the illness. The fever poison seems to expend itself on the different nerve-centres, especially those controlling the respiratory, cardiac, and digestive functions.

1. **RESPIRATORY.**—Bronchitis is an invariable accompaniment ; it lingers long after fever has gone, and may not finally disappear till weeks after. Pneumonia is a frequent sequel in neglected cases, or it may mark the very onset of the disease, and destroy life at once. Tracheitis and laryngitis are also

frequently present, and often from the initial stages. During convalescence a persistent cough may be present, although auscultation reveals no physical signs. It will be found, however, that there is sufficient cause in the inflamed condition of the upper air-passages.

2. **CIRCULATORY.**—Pericardial or endocardial inflammation, as in acute rheumatism. Syncope, from sudden failure of heart's action.

3. **DIGESTIVE.** Tonsillitis, vomiting, diarrhoea, muc-enteritis, and gastro-intestinal catarrhs.

4. URINARY.—Hæmaturia, albuminuria, vesical catarrh.

5. NERVOUS.—Various neuralgiæ ; peripheral neuritis ; mental depression, at times amounting to melancholia ; loss of memory ; and amnesia.

6. SPECIAL ORGANS.—Ear. Otitis, both external and internal, frequently followed by perforation of membrana tympani.

Treatment.—1. PROPHYLACTIC.—The great preventatives appear to be fresh air, plenty of good food, and freedom from overwork and anxiety. In view of its established infectious character, persons should refrain from visiting houses or localities where the disease is present. Eucalyptus, quinine, antipyrin, salicin, and other remedies have been advocated as preventatives ; but we have no actual proof of the efficacy of any in this direction.

2. CURATIVE. Keep the patient isolated in bed, and in a warm room well ventilated. The diet should consist of hot non-alcoholic drinks, eggs, milk, and beef-tea. Oranges may be allowed freely, as they appear to satisfy a great craving. As regards drugs, we have found excellent results from the following broad line of treatment, viz. give a purge at the onset (calomel gr. iij. is best) ; follow this by salicylate of soda (gr. x.) and bromide of ammonium (gr. x.) every fourth hour. The patient's pains are relieved, and he has refreshing sleep, and there appears to be less tendency to pericardial and heart complications. Indeed, the fever and the results of its treatment bear, in many respects, a resemblance to acute rheumatism. Complications of heart and lungs, &c., should be treated on usual grounds. When the fever has subsided the patient will probably require good nourishing food and, if possible, a generous wine (Burgundy, Champagne) or good malt liquor. Convalescence will be hastened by tonics, cinchona, nux vomica, iron, cod-liver oil. A complete change of air to the seaside effects wonders in removing cough. For various other methods of treatment, see 'Brit. Med. Journal,' February 1892.

DIPHTHERIA (MEMBRANOUS CROUP)

Definition.—A specific, contagious disease, characterised by the formation on the mucous membrane of the throat, air-passages, and elsewhere, of a false exudative membrane ; also by marked exhaustion, anaemia, and other grave constitutional symptoms.

Causation.—(a) **PREDISPOSING CAUSES.**—*Age.*—Especially prevalent in children under ten. *Climate and Locality* appear to exert little or no influence. It prevails in hot as in cold, damp climates ; but more frequently in spring and early summer. It occurs in towns and on the country-side. *Family Predisposition.*—Certain families appear prone to acquire diphtheria, as also scarlet and other specific fevers, and in them the mortality is high. *Station of Life.*—Poverty, squalor, unsanitary dwellings, are strong predisposing factors. *Parturition.*—The lying-in woman has a special tendency to receive the disease.

(b) **EXCITING CAUSE.**—A specific poison given off by the breath or exudative material. Loeffler's bacillus is now regarded as the real exciting cause. The bacilli are rod-like bodies the same length as a tubercle bacillus, but much thicker, with here or there swollen or club-like ends. When they are applied to the air-passages of rabbits and fowls they produce the characteristic false membrane ; but it is necessary that the mucous membrane should be in an unhealthy condition, or present small abrasions or injuries.

The disease may be imparted by the sputa, or breath, or exhalations of a patient suffering from the disease. It may also be conveyed by fomites (clothes, bedding, &c.). Evidence tends to show that the disease is primarily a local one (an inoculation), which, extending, causes constitutional symptoms. It is eminently contagious and infectious. It often prevails in epidemics which may be local or widespread, and in which the mortality is high. Sporadic outbreaks, on the other hand, are frequent, when the death-rate is not so marked. It may originate *de novo* in dwellings in close proximity to cess-pools, or to ashpits and middens where decomposing fish or

other animal matter is allowed to accumulate. The micro-organism is apparently thus cultivated, although no previous case of diphtheria had existed.

Incubative Period.—Two to five days.

Symptoms.—The disease varies much in the intensity and character of its symptoms. It may be a mild disease only ; or it may give rise, by the abundance of the false membrane, to grave mechanical obstruction to respiration ; or the attack may (as in scarlet fever) assume a malignant character from the onset, the patient dying early from blood-poisoning. In the same epidemic the symptoms in one case may be of the mild variety, and in the next of the malignant form. This appears to be due not so much to the virulence of the poison as to the receptivity, or to the constitutional weakness of the sufferer.

In an ordinary case the symptoms commence with some abruptness. At first a general feeling of 'out of sorts' is succeeded by anorexia, feverishness, pain and dryness in the throat ; the face is pallid or heavy looking ; the pulse soon rises to 100 or more, and is feeble in character. Respiration is shallow, quickened, and diaphragmatic. The temperature is raised (100° to 102°), but, as a rule, there is not so much fever as the severity of symptoms would suggest. On inspecting the throat, the mucous membrane over the soft palate and tonsils is red and congested.

After twenty-four hours a greyish white spot appears, generally on one tonsil, or it may be on the soft palate. The tonsils are slightly cedematous, the arch of the palate is swollen, and the cervical glands enlarged and tender. Soon other points of infection appear, the spots then increasing in size and becoming greyish in colour, with a tendency to coalesce. The bowels are constipated ; the urine is scanty, occasionally smoky from the presence of blood, and contains albumin almost from the first. There is great prostration from the onset, the pulse is feeble, and the heart-sounds weak. Pallor is also a marked characteristic, and is due to exhaustion and the effect of the poison on the system, and not to the albuminuria.

On or about the fourth day, if the case be a mild one, a general improvement takes place. The pellicle softens and becomes detached and is expelled ; the pulse improves in tone ; the cervical glandular enlargements subside ; food is more easily taken ; and the temperature falls often below the normal line.

But, on the other hand, the attack may pass on to the severer 'croupous' form. The symptoms increase in severity. The temperature rises to 103° or 104° ; the pulse rate increases to 120 or more ; weakness and languor become more pronounced ; there is drowsiness during the daytime, with restlessness and delirium at night. The throat signs are aggravated, swallowing is more difficult or almost impossible, and the voice is hoarse or reduced to a whisper. The mucous membrane of the throat generally is livid and œdematous, the tonsils and lymph glands increase in size, whilst the pellicle extends over the soft palate from pillar to pillar. The fauces are also covered with a tenacious mucus, and there is pronounced fœtor. About the end of the first week the disease has probably extended to the air-passages, causing lividity, stridor, and dyspnœa. The urine is more scanty, more albuminous, and contains hyaline and blood casts. At this stage even, the membrane may soften, become detached and coughed up, with immediate amelioration of symptoms.

If the disease assume the malignant type, symptoms of septicæmia supervene. The fœtor of breath increases ; the false membrane turns brown and softens ; there is an ichorous discharge from the nostrils, excoriating the mucous surfaces on which new pellicle forms ; brawny swelling involves the neck from the parotid regions almost to the clavicles ; the face is bloated ; the pulse feeble and intermittent ; the temperature falls ; and the patient usually passes on to collapse and death, recovery under this condition being rare.

The false membrane consists mostly of exuded fibrin, with proliferated cells on the surface containing micrococci ; whilst deeper there are spores, the deepest layer consisting mainly of blood cells and leucocytes.

Complications and Sequelæ.—1. **EXTENSIONS.**—To trachea

and bronchial tubes generally (suspected by hoarseness and lividity) ; to pharynx ; to Eustachian tube.

2. HÆMORRHAGES.—From tonsils, fauces, or air-passages ; subcutaneous ecchymoses.

3. ABSCESSSES.—In the planes of areolar tissue of the neck ; or in the mediastina.

4. CUTANEOUS.—Formation of diphtheritic membrane on skin at some ulcerated surface, or on vulva or anus.

5. RESPIRATORY.—Stenosis of trachea ; bronchitis, pneumonia, collapse of lungs.

6. MUSCULAR PARALYSES.—Due to neuritis, involving the muscular tissue of the soft palate (food returned by nose), pharynx (choking), eyeballs (strabismus), extremities, heart (syncope or sudden death), diaphragm (dyspnœa), ciliary muscle (loss of accommodation), and neck (rare). A numbness of extremities, especially of arms, may be complained of all through subsequent life. The knee jerks are usually lost.

Diagnosis.—1. FROM SCARLET FEVER.—This fever is known by the rash, by extension of the throat-inflammation to the pharynx, by the absence of albumin in the early stages, and by the subsequent tendency to dropsy.

2. FROM ACUTE TONSILLITIS.—In this affection the invasion is sudden, and runs a quicker course. The inflammation may go on to suppuration (quinsy) ; further, pallor and exhaustion are not marked features ; there is no false membrane, nor are the cervical glands involved. It is usually also one-sided : but so may diphtheria be. If, however, there is on one tonsil a membranous patch which tends to increase in size and to invade the soft palate, and which is accompanied by hoarseness and other signs of laryngitis, the case should be regarded as more than suspicious of diphtheria.

3. FROM THRUSH.—The aphthous patches appear as round points, on the cheeks and tongue by preference ; and they are of acid reaction, and flourish in acid secretions.

Prognosis. A very fatal disease. In some epidemics the death-rate has risen to 80 per cent. or higher. Frequently whole families have been wiped out. It is especially fatal in the young and feeble ; although infants at the breast may

escape the disease. Gravity of prognosis is increased by extensions, especially to trachea and air-passages; by slowness and feebleness of pulse; by fall of temperature during the height of the disease. Death may be due to asthenia, to asphyxia, or to syncope (cardiac paralysis). Hence death is liable to supervene, even in apparent convalescence, from straining at stool, or any muscular exertion.

Treatment.—The patient should be placed in a warm room, the temperature of which should be maintained at 60° by means of a good fire and a steam-kettle. An atmosphere of sulphurous acid, procured by placing sulphur on live coals, is always well tolerated by the patient, and exerts a beneficial influence.

All vessels, utensils, and linen used by the patient should be carefully cleansed and disinfected, or destroyed.

1. LOCAL.—The local treatment consists of the use of various gargles or the application of washes or sprays. Few diseases have had so many local remedies. As gargles we may give tincture of perchloride of iron (℥xxx. ad ℥j.), or nitrate of silver (gr. v. ad ℥j.), or solution of free chlorine. Dr. Donald Hood recommends that all gargles should be warmed. We may apply solutions of permanganate of potash (gr. ij. ad ℥j.), of carbolic acid (1 in 25), or sprays of solution of sulphurous acid, lactic acid, etc. We have also seen good results from swabbing the throat with papain, with solution of chloride of zinc (20 per cent.), or better still with saturated solution of salicylate of soda. Sprays of solution of boracic acid (gr. iij. ad ℥j.) are also useful. Steam inhalations may also be ordered with advantage; and the occasional allowance of ice to suck is a grateful adjunct.

2. CONSTITUTIONAL.—The disease is one which urgently requires plenty of good food. It should be in concentrated liquid form, such as beef-tea, chicken broth, and other animal food extracts, eggs, milk. Stimulants also are generally demanded, the quantity being regulated by the condition of the pulse. For insomnia, bromides are the safest; yet morphia may be given, if carefully watched. Avoid constipation, owing to the risk of fatal syncope when straining at stool.

The question of tracheotomy when asphyxia is threatened is an important one. It should be performed at once if danger be imminent, a delay even of a few hours being, at times, disastrous. It is a good rule, however, not to perform the operation on infants under one year of age, as it is seldom successful, nor should it be advocated if there are other conditions which point to impending death. Intubation of the larynx has been successfully practised; and in many instances appears to be more successful than tracheotomy. (See W. W. Ord, 'Thesis for M.D. Oxon.')

During convalescence, which is often prolonged, iron, quinine, cod-liver oil, or other tonics are indicated.

MALARIAL FEVERS

Definition.—Fever which are due to the reception into the blood of specific poisons emanating from marshy, uncultivated lands; and characterised, for the most part, by attacks which are paroxysmal, with complete intermissions between; or by a general febrility to which, from time to time, distinct exacerbations are added; these exacerbations remitting only, the actual fever not terminating with the remission.

Causation.—(a) **PREDISPOSING.**—*Age* and *Sex* appear to exert no real predisposing influence. The disease occurs more frequently in adult males, but this is probably due to their greater exposure to malaria. *Climate.*—More frequent in temperate or in tropical countries; yet the fever is not due to heat alone, which is only one predisposing factor. *Race.*—More prevalent amongst Europeans than dark races; and fresh comers to a district where the disease is endemic are particularly prone to acquire it. *Locality.*—Low, damp regions. Still it may prevail in places—*e.g.* a row of houses—which are elevated, but exposed to winds which have passed over malarial ground. *Condition of Life.*—Privations, fatigue, all predispose; as also does a *previous attack*.

(b) **EXCITING CAUSE.**—A specific organism. The generation of the poison is not due to wet, as it frequently disappears

in malarial regions during the rainy season ; nor is it due to vegetable decomposition, else London, with its dust-bins, would suffer ; but it appears to be dependent on two or three factors, the principal being a porous, damp soil, rank vegetation, and want of cultivation. Swampy land when drained and cultivated no longer gives off the miasm ; but if allowed to relapse into its primitive state, ague again prevails. Nevertheless, drainage in some instances increases the poison, this being due to the exposure to heat, and the decomposition of vegetable matter which had been previously covered by water. For similar reasons an outbreak of ague may result from excavations or other disturbances of soil, the poison which had been buried, maybe for years, being thus disturbed and liberated. Thus coolies in India contract ague when making railways and breaking up the soil.

The miasm is confined, as a rule, to low-lying ground, and does not extend to any great altitude ; it easily passes along the surface from one region to another, but it is absorbed by running water, and its spread is impeded by a belt of trees. Possibly the same poison, or the miasm from the same swamp, may produce dysentery.

Laveran has described a specific hæmatozoon of spherical, or flagellate, or crescentic shape, which attacks the red blood corpuscles and lives at their expense.

INTERMITTENT FEVER (AGUE)

Definition.—A specific, non-contagious fever due to malaria, occurring in paroxysms which are characterised by a cold, a hot, and a sweating stage ; with intervals of comparative good health between the attacks, and a tendency to recurrence.

Incubative Period.—Uncertain. Probably from six to twenty days.

Pathology.—The spleen is deeply engorged with blood, and consequently firm, and enlarged to many times its natural size. A similar engorgement is found in the liver, and in the submucous tissue of the alimentary canal generally. The blood

itself is thin and pale, from increased quantity of leucocytes, and dropsical effusions are consequently frequent in severe cases.

Symptoms.—In people who have suffered previously there are some premonitory signs by which an oncoming seizure may be recognised, such as languor, headache, constipation. If the temperature be taken, it will be found to have risen to about 100° .

But in a first attack the typical symptoms commence at once. Its course is divided into three distinct stages: the cold, the hot, and the sweating.

COLD STAGE.—Commences with drowsiness and headache followed by rigors, tremblings of limbs, chattering of teeth, 'goose-skin,' and a general sense of cold, especially in the loins and back. The face is pallid, the hands blanched, the nails being blue or livid. The patient asks for blankets and warm clothing, yet the actual temperature of the body is febrile, being as high as 102° or 104° . The pulse is quick and small; the urine copious and pale, with an increase of urea and uric acid. This stage lasts about an hour, and is followed by the **HOT STAGE**, during which the chilliness gives way to a sense of comfortable warmth; the aspect of the patient is also changed, the face being flushed, the conjunctivæ injected. The skin becomes moist; there is marked thirst; the pulse is increased in volume; the lumbar pain is diminished, and is replaced by severe headache; the urine is still abundant, but darker; the respirations shallow and quick; and the temperature rises often to 105° or 106° , although it is never so high as the patient's symptoms would lead us to suppose. On examining the abdomen the spleen is found enlarged and softened, and the liver painful. This stage lasts about two hours. To this succeeds the **SWEATING STAGE**.—Here the temperature falls; the skin becomes moister; then a copious diaphoresis occurs, first on the forehead and chest, and then gradually over the whole body. The general pains disappear, the respiration becomes easy, the urine is scanty and loaded with lithates, although the urea is diminished. Gradually the sweating subsides, and the patient falls into a refreshing sleep. This stage lasts about an hour.

In different attacks all the various stages may be shortened, the whole seizure being completed in from two to three hours ; or an attack may be anticipated or postponed by some surrounding influences. During the interval the patient is apparently well, though he may suffer from dyspepsia or giddiness and other discomforts. In India there does not appear to be any distinct type of ague. A case does not present the cut-and-dried symptoms described in books ; but there is usually a daily exacerbation of fever in those who suffer from ague.

Varieties.—1. **QUOTIDIAN AGUE.**—The commonest variety, in which the attack occurs daily at the same hour, most frequently at about ten A.M.

2. **TERTIAN.**—In which forty-eight hours intervene between the first symptoms of the seizures. Thus it recurs every third day.

3. **QUARTAN.**—In which seventy-two hours elapse between the outbreaks, which recur, therefore, every fourth day.

4. Other subvarieties are described (**DOUBLE QUARTAN**, **DOUBLE TERTIAN**, &c.) which may be regarded as mixtures of quotidian ague and one or other variety. The nomenclature, however, is clumsy throughout.

Diagnosis.—1. **FROM PYÆMIA.**—By the history of injury or surgical operation, the odour of patient, the absence of marked splenic enlargement ; finally, quinine has no appreciable effect.

2. **FROM ULCERATIVE ENDOCARDITIS.**—By the signs of extensive valvular disease, cardiac hypertrophy, and a rheumatic history. The febrile intermissions are also more irregular and extensive.

Complications and Sequelæ.—1. **RELAPSE**, or return of the disease at some future period, even though the patient be removed from the aguish district. 2. **PERIODICITY** in character of other subsequent ailments (neuralgia, pneumonia, typhoid fever, &c.), in which the symptoms common to any acute disease are aggravated on certain days, or at certain hours. This is especially seen when the ague was mild and not a typical attack. 3. **CONDITIONS OF BLOOD.**—The red cells are dimi-

nished in quantity and broken up, the fibrin is lessened, and the blood pigment deposited in the different tissues, notably in the skin, producing a characteristic dirty pallor. Hæmic murmurs are often heard at one or other of the cardiac orifices. 4. SPLEEN becomes firm and enlarged, often to an enormous extent ('ague cake'). 5. GASTRIC DISTURBANCES.—Impaired appetite and digestion, diarrhœa. 6. RESPIRATORY.—Bronchial catarrhs, capillary bronchitis, pneumonia. 7. NERVOUS. Patients are apt to lapse into despondency, or marked hypochondriasis.

Treatment.—(a) PROPHYLACTIC. — In malarial districts select for residence an elevated site with, if possible, a belt of trees or a running stream intervening between it and the swamp. Avoid exposure, especially in the evenings. Drinking-water should be effectually filtered or, better still, boiled. Give small doses of quinine (gr. ij. three times a day).

(b) CURATIVE.—Quinine is the great specific remedy. It is best given in one large dose (gr. x. to gr. xv.) at the end of the cold stage. Some prefer doses of five grains every two hours all through the attack. The efficacy of the drug is enhanced by a preliminary purge. In some rare instances quinine fails; we may then fall back on arsenic, beginning with *m̄v.* of the Fowler's solution every three hours, and increasing the dose as the drug is tolerated. It appears, however, to be more efficacious in the chronic form of the disease, and as a blood tonic during convalescence, especially when the spleen is enlarged and indurated. Often a course of waters (Homburg, Woodhall Spa) will have beneficial results in restoring to health. Occasionally we may resort to the preparations of salicin in cases where quinine disagrees. Warburg's tincture has a justified reputation in India. Give a table-spoonful three times a day.

REMITTENT FEVER

Definition.—A specific fever due to malaria, characterised by exacerbations occurring in paroxysms which remit, but in which there is no distinct intermission. It is the commonest form of malarial fever in tropical climates.

Pathology.—Similar changes are observed in the spleen, liver and intestines as are found in intermittent fever.

Granules of varying shape, but mostly of ovoid form, and containing pigment, have been described as occurring in the blood, lymph-glands, spleen, and capillaries.

Symptoms.—The disease is mainly characterised by general fever with paroxysmal exacerbations. The onset is sudden. As in intermittent fever, there are three distinct stages, viz. the cold, the hot, and the sweating.

The COLD STAGE is short, and hardly perceptible; the temperature, however, being slightly raised. The HOT STAGE lasts from six to ten hours. It is accompanied by very high fever (105° to 107° , or even higher), a pulse rate of 120, quickened breathing, flushed face, pungent, dry skin, a foul tongue, and general pains in the limbs and back. The urine is scanty and high coloured. In addition, stomach signs are an important feature. The patient complains of epigastric tenderness and distress; vomiting is severe and urgent, and is frequently complicated by hæmatemesis or by jaundice. The SWEATING STAGE is imperfect, attended only by slight diaphoresis; the headache, nausea, and vomiting diminish; the temperature falls slightly, but does not reach the normal line. The fever is thus said to remit, the length of the remission varying from an hour or two, to ten or twelve hours; the longer the remission the more favourable the prognosis.

The paroxysms occur two or three times in the twenty-four hours: they increase in severity, or at least are more marked during the first few days of the attack; and altogether the disease is a more serious one than intermittent fever. It lasts from one to two weeks.

Varieties, named chiefly from the prominence of certain symptoms. Thus, when epigastric distress and tenderness, hæmatemesis, and jaundice are the most pronounced characteristics, we speak of *Bilious Remittent Fever*. Another variety is distinguished by the advent of high fever of a 'typhoid' type, with low muttering delirium, or by wild mental excitement followed by coma and death.

Diagnosis.—1. FROM YELLOW FEVER.—In this disease,

hematemesis ('black jack') is always present ; and albuminuria is the rule. Quinine has no specific effect ; and, although eminently contagious, the disease is seldom imported to latitudes where the mean temperature is not above summer-heat.

2. FROM ENTERIC FEVER.—This fever may be complicated by symptoms distinctly pointing to malarial infection ; then the diagnosis is difficult. In ordinary enteric fever we are guided by the presence of rash, by the gradual and slow rise of temperature to the climax—which is not reached till at least the end of the second week—and its equally gradual decline. Quinine, also, has no appreciable effect.

Treatment.—Quinine is the specific remedy. It may be given in small doses (gr. iij. every four hours) all through the fever, or in larger quantities (gr. x. to xx.) on the first indication of remission. If the stomach cannot retain it, administer the drug by enema or by subcutaneous injection. A preliminary purge (Calomel, gr. iv.) is beneficial. Aconite has been very warmly advocated of late years. Vomiting should be allayed by effervescent salines ; but this symptom diminishes as quinine begins to take effect. The patient's strength must be maintained by good food, and by stimulants, if necessary.

ERYSIPELAS

Definition.—An acute, specific disease, eminently contagious in the presence of wounds, characterised by inflammation of the skin and subcutaneous cellular tissue, and attended by severe febrile symptoms.

Causation.—(a) PREDISPOSING.—*A sore, or open wound.* The uterus after parturition comes under this category. It is stated that it invariably occurs in relation with some wound or abrasion of surface—a statement which must be accepted with caution. *Age.*—It may occur at all periods of life, but especially in infancy, and after forty years of age. *Constitutional Defects*, such as those due to chronic alcoholism, exhaustion, exposure, and a depraved life. Further, some people, from one or other unknown cause, seem consti-

tutionally prone to contract the disease. *Season of Year*.—Winter and spring, during the prevalence of cold and east winds. *A Previous Attack*.

(b) **EXCITING CAUSE**.—A specific micro-organism.

Dr. Fehleisen has described a micrococcus which is always present in erysipelas, and which produced typical erysipelas when introduced directly into the blood of rabbits. The micrococci appear to grow in chains, and occupy principally the lymph spaces and channels of the skin.

Incubative Period.—Not known; probably about four days.

Symptoms.—In the idiopathic form, which we can only consider here, the onset is sudden, accompanied by chills, rigors and other signs common to specific febrile states. This stage usually lasts about two days, but may be shorter. The typical inflammation of the skin commences usually, but not necessarily always, on the face, at the junction of the nose with the cheek, or on the external ear, as a red elevated spot, whence it extends, as from a centre. The skin over the affected area becomes red, tense, swollen and œdematous. The edge of the blush is raised, and spreads by irregular outline across the middle line of the face, or to the eyelids, the scalp, the external ear, lips, or other locality. Hence, the face and physiognomy become altered almost beyond recognition. Bullæ containing turbid or purulent serum occur in severe cases. After a time, although the blush started from a point and extended centrifugally, it shows a tendency to creep in one direction, whilst it dies away in the opposite. The eruption, however, is frequently arrested where the skin is closely attached to some bone or fibrous tissue. At the same time the constitutional symptoms are severe. There is high fever, the temperature reaching 102° to 105° ; the pulse-rate is accelerated to 120 or more; the tongue is foul, coated with a thick white fur; the bowels are constipated; the urine is scanty, often albuminous, and contains a diminished amount of chlorides. In severe cases there is a tendency, especially at night, to delirium, which may be of a low muttering type in severe cases.

About the sixth day the temperature falls somewhat suddenly, as if by crisis ; the colour of the blush fades ; the tenderness and swelling diminish, and are finally followed by desquamation. The crisis is often attended by diarrhœa, or violent perspiration, or severe delirium.

On the other hand, instead of the fever abating, the symptoms may become unfavourable. Delirium increases, there is subsultus, the tongue becomes brown and dry, sordes form on the lips and teeth, the pulse becomes irregular, and the patient's condition passes into the 'typhoid' state, which is terminated by coma and death.

Complications and Sequelæ.—1. CUTANEOUS SYSTEM.—Sloughs, abscess, and resultant pyæmia.

2. URINARY SYSTEM.—Acute tubal nephritis.

3. RESPIRATORY SYSTEM.—Extension of the inflammatory process to the larynx, causing œdema of the glottis ; pneumonia ; pleurisy.

4. CIRCULATORY SYSTEM.—Phlebitis, embolism.

5. NERVOUS SYSTEM.—Severe delirium ; meningitis, by extension through the ophthalmic and other veins of the skull.

Diagnosis.—The disease may be simulated by one or other skin affections which, from some local cause or irritation, are attended by more inflammation than usual—e.g. *Acne Rosacea*, *Alcoholic Erythema*—but in each case the temperature and the history form sufficient guides. In severe *Herpes* the surrounding erythema may also resemble erysipelas, but the affection is limited to certain localities in the course of cutaneous nerves.

Prognosis.—As a rule, favourable. It is, however, a grave disease in debilitated and exhausted patients, in alcoholic subjects, and in parturient women.

Varieties.—Many subvarieties have been artificially named, according to the intensity of the local inflammation. Thus we speak of *E. Simplex*, in which the fever is slight only, and accompanied by little more than an erythema of skin. If the subcutaneous areolar tissue be involved, it is known as *Edematous Erysipelas* ; whilst that form which is

attended by the formation of pus and sloughing of deep tissues is called *Phlegmonous Erysipelas*.

Treatment.—(a) **GENERAL.**—Isolate the patient. A preliminary purge (Calomel, gr. iij.; or Haust. Sennæ Comp.) should be given. The tincture of perchloride of iron is almost universally regarded as a specific remedy. Give large doses (ʒixxx. every four hours), if they can be tolerated. Relieve delirium by chloral hydrate or by bromides. Maintain the patient's strength by good food. Give alcohol if required, the pulse being the chief guide.

(b) **LOCAL.**—The main indication is to prevent exposure to the atmosphere. Thus the eruption may be smeared with zinc ointment or other cooling application; or it may be painted with flexible collodion, extract of belladonna and glycerine, or covered simply by lint or cotton-wool. Flour, though very cool as an application, has the disadvantage of forming crusts with the exudation from bullæ, and then it becomes an attraction to flies and other insects. A mixture of oxide of zinc and powdered starch is, however, extremely useful. Some recommend the application of lunar caustic, either as the solid pencil or in strong solutions (gr. xx. to ʒj.), and in some cases it has certainly appeared to us to arrest the extension of the blush. Or we may constantly spray the face with a solution of Hydrarg. perchlor. (1 in 4000) (Robin). On the whole, however, we have seen the best results from simple lead lotion.

CHOLERA

Definition.—A specific disease occurring, for the most part, epidemically in tropical climates, and characterised by profuse watery diarrhœa, vomiting, cramps in the extremities, and suppression of the secretions.

Incubation.—Not accurately known. It may vary from a few hours to five days.

Causation.—(a) **PREDISPOSING CAUSES.**—*Season.*—It prevails in the hot months from spring to autumn, gradually disappearing during winter. *Condition of Life.*—Insanitary

surroundings, such as are found in badly-drained cities (Marseilles, and the towns of Bengal). The disease also follows certain well-defined tracks taken by caravans, pilgrimages, or the courses of large rivers, or well-frequented roads. *Improper dietary*, such as rotten fruit, decomposed animal food, or other irritants which predispose to diarrhoea. *Soil*.—It prevails in localities in which there is a gravelly or sandy soil, or in low-lying districts. It was the impression of many of the physicians and surgeons sent by the British Government to Egypt during the cholera epidemic of 1883, that the dry, hot sand of the desert favoured the propagation of the disease.

(b) EXCITING CAUSE.—A specific micro-organism. Koch has discovered a well-defined 'comma'-shaped bacillus in the water-tanks from which cholera infection has been supposed to issue, and also in the stools of cholera patients. The bacilli are really short, curved rods connected with each other, thus forming long screw-like threads. Each 'comma,' therefore, appears to be a fragment of a spirillum, averaging 1.5μ in length. The bacilli multiply quickly; they are easily killed by heat, carbolic acid, and bichloride of mercury. (See 'Flügge's Micro-Organisms.' New Sydenham Society.)

It is pretty certain that infection, as in typhoid, is conveyed mostly by drinking water contaminated by cholera evacuations. Further, experiments have proved that the infection is most, or indeed only, active, in decomposing cholera evacuations. Mice fed on food mixed with freshly voided cholera stools suffered no harm, whilst when fed with cholera evacuations which had recently decomposed, they presented all the symptoms of virulent cholera. Murchison also related the case of a drunken soldier swallowing, by mistake, some rice-water stools recently voided, yet with no bad results.

The disease is not necessarily communicable from one patient to another, or to nurses and attendants.

Symptoms.—In a typical attack four stages are usually recognised, viz.: 1. Stage of primary fever. 2. Stage of diarrhoea, vomiting, and cramp. 3. Stage of prostration and collapse. 4. Reaction.

1. *Primary Fever.*—The disease generally commences with

premonitory malaise, nausea, and irregularity of bowels: the condition often being attributed to some error of diet, or to some medicinal irritant. There is some slight febrility, languor, thirst, and colicky pains in the abdomen. This stage is, however, not invariably noticed; indeed, it may be absent altogether, the disease then commencing suddenly with the *Second Stage*, of violent purging, vomiting, and cramps. The evacuations consist, first, of the ordinary fecal contents of the large intestine, followed by a copious biliary discharge; subsequently, the motions assume their characteristic 'rice-water' type, being clear and watery, or turbid from the admixture of flocculent particles of epithelium; they are also of alkaline reaction, and occasionally contain blood. When allowed to settle, they deposit epithelium, mucus, shreds of mucous membrane, and phosphates. Nausea and vomiting are urgent, the vomited matter consisting first of partially digested food, and subsequently of a clear, watery, or whey-like fluid. In addition, the patient complains of great thirst, headache, pains in the epigastrie and right hypochondriac regions, or cramps in the abdomen generally, and in the extremities, especially the legs. 3. The *Collapse*, or 'algide' stage succeeds. The temperature falls rapidly down to 94° or 93° ; the surface is cold, the face is pallid or even ashen grey; the ears and lips are livid; the nose is pinched; the eyeballs sunken and shrivelled; the respirations shallow and hurried, the breath being cold; the voice is hoarse and feeble; the pulse is rapid and, except in the larger arteries, imperceptible. In addition, urine and other secretions are arrested. Nervous symptoms are also prominent, the patient being alarmed and restless with a clear mental condition, or he sinks into a condition of stupor and indifference. When the collapse stage is well-established the vomiting and diarrhoea may cease, and the patient is to all appearances dead, except that he may be roused to slight consciousness. Death may occur in an hour or two, or be deferred twelve hours. On the other hand, recovery is marked by 4. *The Stage of Reaction*.—Lividity disappears; the skin becomes warm, there is some perspiration; vomiting and purging diminish; the urinary and biliary secretions are

again formed; the pulse at the wrist returns, but never exceeds 100; the temperature rises above the normal line; and the respirations become fuller. This stage is not free from danger, as the patient may die from cerebral congestion, or he may sink into a typhoid state and die from exhaustion, with a return of the intestinal symptoms; or he may have fatal pneumonia, or congestions of other internal organs. In women the reaction is not infrequently marked by menorrhagia. This stage lasts from twenty-four hours to three or four days.

Varieties.—Classification based entirely on the severity of symptoms. 1. The disease may be so mild that, notwithstanding vomiting, and diarrhœa with characteristic stools, recovery takes place in twenty-four hours. 2. *Cholera Sicca* is a variety in which there is no vomiting or purging, the patient dying at once, as it were, from the very intensity and shock of the seizure. *Post mortem* the intestinal canal will often be found to contain the characteristic evacuations.

Prognosis.—Always grave. In well-marked epidemics the death-rate is highest at the commencement, when fifty per cent. of cases die; but the mortality is not so high in sporadic outbreaks. The mortality is always higher in the extremes of age, in alcoholic subjects, and in those whose constitutions have been injured by hardships and depravity.

Diagnosis.—In epidemics there is not much difficulty. Sporadic cases may be simulated by the effects of irritant poisons (croton oil, elaterium, arsenic), which cause enteritis; or they may be confused with severe typhoid fever, with perforation of the intestine. In none, however, is the collapse stage so marked as in cholera, nor have the stools the typical rice-water appearance.

Pathology.—The *post mortem* appearances depend on the stage at which death occurs. If during the period of collapse, the most noteworthy feature is an absence of fluids of the body. There is no bile, no serum, the blood being thickened and black, and no urine; the lungs are dry and pale, the arteries contracted, and the intestinal glands swollen; dropsies, and effusions into serous cavities, if they existed before the

illness, will have disappeared. If death takes place during the stage of reaction, the liver, lungs, brain, and internal organs generally, are congested, and the veins, together with both sides of the heart, engorged. Rigor mortis, in most cases, is unusually prolonged.

Treatment.—(a) **PROPHYLACTIC.**—Avoid fatigue, especially in the sun. Check all tendency to diarrhœa, from whatever cause ; a flannel belt round the abdomen seems to exert some preventive influence, probably, however, only by calming the mental anxiety. Food should be plain, nutritious, and digestible ; unsound fruits and decomposing animal food should, therefore, be strictly forbidden. All drinking-water should be kept free from possible infection, and before use should be efficiently filtered, or, better still, boiled. The houses and streets in an infected area should be rigorously cleansed and disinfected. Each patient should be isolated as far as possible. Disinfect and bury deeply all stools and vomited matter passed by patient.

(b) **CURATIVE.**—Various treatments have been advocated, some of which are totally opposed to others in action. Thus, purgative medicines of greater or less severity (calomel, castor oil) have been advised, with a view to cleanse the gastrointestinal tract of irritant matter. Others, again, give astringents from the first, such as catechu, chlorodyne, &c. Warburg's tincture, from its results, has an apparently justified reputation in India. The experience of some of the English surgeons attending the epidemic in Egypt in 1883 was in favour of hypodermic injection of morphia ; or of opium by the mouth, if it could be retained. Dr. Lauder Brunton has given subcutaneous injections of atropine, with a good result.

Relieve the cramps by hot stupes, by morphia subcutaneously, or by opium fomentations, or by mustard poultices. There is evidence that tobacco-smoking also relieves this distressing symptom. For thirst we may order ice, or iced water, if obtainable. Vomiting may be checked by dilute hydrocyanic acid in effervescent alkalis.

During collapse we may order wines or brandy, diluted with warm water, and apply hot blankets. Good results may

be obtained by free (one to eight pints) intravenous injection of a two-per-cent. saline solution at 100° F. The food should consist of milk, eggs, and arrowroot, or other starchy food.

In the stage of reaction the treatment required is the same as in the ordinary febrile states. Ice may be applied to the head. We may also administer saline febrifuges. Milk is best adapted for dietary.

YELLOW FEVER

Definition.—A specific, contagious, continued fever, characterised by jaundice, black vomit, suppression of urine, and a tendency to delirium and coma.

Causation.—(a) **PREDISPOSING CAUSES.**—*Age.*—Persons of all ages equally suffer, if exposed to contagion. *Race.*—The disease particularly affects white people; negroes and dark races are singularly immune. *Station of Life, and Surroundings.*—As in typhus, yellow fever is favoured by overcrowding, insufficient ventilation, deficient and defective food. It is therefore prevalent in cities, especially in the lower and poorer parts, and in densely populated houses contiguous to those harbours where garbage is allowed to decompose under a tropical sun; indeed, the epidemic may be limited to certain parts of a town or seaport. New comers to an infected area are especially prone to contract it.

(b) **EXCITING CAUSE.**—A specific poison, which is probably received into the system by the respiratory tract. It may be conveyed from one to another by fomites, such as clothes, bedding, and other media. For the development of the poison a mean temperature of 70° F., or higher, is necessary. The fever, therefore, is essentially a tropical one, prevailing, except in rare instances, between the latitudes of 45° N. and 35° S. If the fever should break out on board ship, it will generally have disappeared after the vessel has passed these boundaries. Still, cases have been imported into Swansea and other harbours of South Wales, but the disease did not extend.

Incubative Period.—Two to fourteen days.

Pathology.—The face is bloated and jaundiced ; decomposition sets in early. There are congestion and catarrh of the gastro-intestinal tract as far as the ileo-cæcal valve. Ecchymoses and petechiæ are common in the stomach, œsophagus, lungs, and pleura. The liver is enlarged, yellow-stained, and fatty ; or, if death occur in the later stages, it is shrunken. The heart and kidneys are in a state of fatty degeneration.

Symptoms.—Those of a continued fever, somewhat resembling typhus. The invasion or premonitory fever is sudden, the patient occasionally being seized when at work, or even when asleep. It is characterised by rigors, headache, pains in the limbs and back ; the face is dusky or drunken-looking, and the conjunctivæ are injected ; the urine is diminished ; the bowels are constipated ; and the tongue is heavily coated. The bodily temperature rises at once, often reaching as high as 102° on the first day.

On the Second Day the fever is accompanied by epigastric tenderness and by vomiting, which becomes rapidly urgent, the vomited matter at first consisting of the partially digested contents of the stomach, then becoming clear and watery, and, finally, black and bilious (black vomit, 'black jack'). At this stage there is a fall in the pulse, often below normal, as if from collapse, although the temperature has risen considerably (104° to 105.5°).

On the Third Day the fever still increases ; the temperature may even rise to 107° – 108° , or higher. There is marked jaundice, the skin and conjunctivæ being yellow ; the urine is scanty and bile-stained ; the gums are spongy, sore, and easily bleed ; hæmorrhages also occur from the buccal cavities and nose ; and a petechial eruption is present in the skin.

On the Fourth Day, or on the fifth at latest, the patient improves. The temperature falls to normal, or lower ; the pulse-rate sinks to between 40 and 50 ; the tongue begins to clean ; vomiting ceases ; jaundice diminishes ; and the case may proceed to convalescence, which is protracted.

A relapse, however, may occur. The fever then returns, with jaundice and hæmorrhages as before, but more intense than ever, accompanied by delirium, collapse, uræmic con-

vulsions, and coma. Recovery even at this stage is not unknown.

Varieties.—Described according to the intensity of the symptoms. An attack may be so mild as to present signs of only a temporary bilious disorder ; or, on the other hand, the patient may die on the first day from virulent jaundice accompanied by coma. Further, in certain cases there is no order of symptoms as detailed above ; in some, hæmorrhage occurs early, and is fatal ; in others, suppression of urine is the most pronounced feature, and is equally fatal.

Diagnosis.—As a rule, easy, when we remember its local outbreak in tropical regions. It may be confounded : With 1. *Acute Atrophy of the Liver* ; but in this disease the onset is gradual and without epigastric pain, or black vomit. 2. With *Small-Pox* of malignant type, in which the characteristic eruption is our chief guide.

Prognosis.—Always grave, especially to Europeans. It is mainly influenced by the patient's temperature. An observation of 106° is usually followed by fatal results, death being due, apparently, to the effects of hyperpyrexia.

Treatment.—(a) **PROPHYLACTIC.**—Hygienic measures are most important. Destroy all refuse and decomposing matter. Cleanse and disinfect the house or ship in which a case occurs. Enforce strict isolation and quarantine, and prevent immigration of healthy people into the infected area.

(b) **CURATIVE.**—The patient should have a cool, well-ventilated room, allowing at least 2,000 cubic feet of atmosphere, if possible. Diet should consist of milk, beef-tea, farinaceous foods, and other non-irritating sustenance. In view of the irritable condition of the stomach, nourishment should be given in small but frequently repeated quantities. There is no specific remedy. Allay vomiting by iced drinks, by sinapisms, or by chloroform applications to the epigastrium, or by hypodermic injections of morphia. Promote diuresis by hot air baths or packs ; this will not only help to relieve the kidneys, but will bring down the temperature. The suppression of urine would make us careful in the use of cold packs, whatever the amount of hyperpyrexia. We may use,

instead, cold water injections into the bowel, or give quinine or aconite. Purgatives (calomel, castor oil) are recommended in the early stage. Notwithstanding the restlessness and the tendency to collapse, opium and alcohol should always be given with caution.

CEREBRO-SPINAL FEVER (EPIDEMIC CEREBRO-SPINAL MENINGITIS)

Definition.—An acute, febrile disease occurring mostly in epidemics, and characterised by delirium, pains in the back, general hyperæsthesia, and other symptoms pointing to an inflammatory condition of the membranes of the brain and spinal cord.

Causation.—(a) **PREDISPOSING CAUSES.**—*Age and Sex.*—It affects children and young adults, especially healthy young men. *Season.*—Epidemics prevail most frequently in winter or a cold, bleak spring. 3. *Station of Life, and Surroundings.*—The fever is prone to attack the poor, and those who are exposed to fatigue from excessive exertion, privations, and consequent overcrowding.

(b) **EXCITING CAUSE.**—No definite micro-organism has been discovered. The disease prevails in epidemics, not so much widely spread as in scarlet fever, or influenza, but in scattered foci. It appears to be eminently infectious.

Pathology.—The principal changes are found on the surface of the brain, spinal cord, and in their membranes. In the early stages the pia mater is in an inflammatory state, being sticky and adherent from fibrinous exudation; there is an increase of the subarachnoid and intraventricular fluids; and superficial hæmorrhages are not uncommon. These changes are found all over the surfaces of the brain and cord, but are specially marked at the base of the brain and at the points of exit of the cranial and spinal nerves. In the later stage the exuded inflammatory products become turbid, or green and purulent. The lungs, liver, and spleen also are not unfrequently congested; probably, however, as a result of the general febrile state.

Symptoms.—The onset is generally sudden, being marked by fever, rigors, severe headache involving the frontal or occipital regions or both ; by frequent vomiting without premonitory nausea ; and by pains in the whole length of the spinal column and muscles of the back, extending to the limbs. As the disease progresses the fever increases. The tongue is coated ; the bowels are constipated ; the temperature rises to between 100° and 104° , or even higher ; the pulse is slow and irregular, and bears no relation to the height of the temperature ; the breathing is shallow and sighing ; there is acute hyperæsthesia of the skin, and tonic contractions of the muscles of the back (opisthotonos). Cutaneous eruptions of various characters are also occasionally met with : they may be vesicular, or papular, or petechial and black. The patient is irritable, but soon becomes drowsy and delirious. According as the various special nerves become involved at their exit from the cranium, we may find strabismus with inequality of pupils, deafness, or tinnitus, &c. Death may ensue in forty-eight hours from collapse, owing to the intensity of the inflammatory attack ; or it may be deferred six or eight days, being then due to coma or to severe complications. Various types of the fever have been described, according to the intensity of different symptoms. A relapse, after apparent convalescence has set in, occasionally occurs, and is generally fatal.

Complications and Sequelæ.—Mostly due to trophic changes in nerves—*e.g.* sloughing of cornea, complete and permanent deafness, bedsores and gangrene, paraplegia, or paralysis of a limb or of a group of muscles, neurotic dystrophies of joints.

Diagnosis.—1. FROM TETANUS.—In this disorder the contraction of muscles is unattended by any rash, and there would be a history of some traumatism. 2. FROM PURPURA.—Here we are guided by the history of the case, and by the absence of any marked fever and nervous symptoms. 3. FROM TYPHUS FEVER, which is not attended by muscular convulsions, nor by widely distributed pains. Also, the rash of typhus, although preceded by a papular stage, is soon

petechial. Nor has epidemic meningitis the characteristic odour of typhus fever.

Prognosis.—Should always be guarded. The mortality of all cases is quite fifty per cent., being especially high in young children. Coma, convulsions, and relapse are conditions of extreme gravity.

Treatment.—We can only apply remedies with a view to relieve the pain and the effects of the meningeal inflammation. With this object we may order leeches or blisters to the nape of the neck, or along the spinal column; or apply ice (Chapman's bags). Quinine (in large doses, gr. v.) may be given to lower temperature. Subcutaneous injections of morphia are indicated, except in infants, to relieve the intense pain; or we may give bromide of potassium (grs. v. to xx.), hyoscyamine (gr. $\frac{1}{100}$), and other sedatives. Stimulants should be given with care, owing to the congestion of the nervous centres. In convalescence, iodide of potassium will be of some value in absorbing the products of inflammation.

DYSENTERY

Definition.—A specific, febrile disease, characterised by inflammation of the mucous coat and glands of the large intestine, accompanied by diarrhœa and tenesmus.

Causation.—(a) **PREDISPOSING CAUSES.**—*Age and Sex* apparently exert no predisposing influence, beyond that adult males are more exposed to infection than others. *Climate and Season.*—It prevails in malarial regions, in countries possessing variable climates, and during summer and autumn. *Locality.*—In such countries where the disease is endemic, it exists especially in damp, marshy, low-lying districts; or where the water-supply is contaminated by drainage. *Conditions of Life.*—It is predisposed to by damp, exposure, bad food, privations (war); also by various effluvia emanating from unhealthy wounds, or from decomposing matter. *Errors of Diet.*—Salt meats, and other indigestible food (especially in patients who have been previously attacked); also copious libations of cold water will bring on an attack.

(b) **EXCITING CAUSE.**—Probably some specific micro-organism ; but little is as yet known. The evacuations appear to be most infectious, and especially after their decomposition. In the tropical form of dysentery they contain the *Amæba coli*, which is thought to be the exciting cause.

Pathology.—The chief lesion is confined to the large intestine, especially the lower colon, sigmoid flexure, and rectum. The mucous coat is generally congested ; the solitary glands are enlarged ; the epithelia lining the crypts of Lieberkuhn are proliferated, and with the exuded inflammatory products, fill these follicles, and are subsequently shed in the form of casts, as in the renal tubules. As the inflammatory process advances, there is destruction of tissue, either in isolated patches, or in larger areas ; or the ulceration may extend under the mucous surface, and so connect different sloughing foci, leaving connecting bridges of apparently normal tissue between. In advanced cases the submucous coat becomes hypertrophied ; the ulceration may extend to the muscular, or even the serous tunics, and cause perforation. If recovery takes place, contraction of the various coats, especially of the fibrous, frequently causes narrowing and stricture.

Other pathological changes may exist, notably hepatic abscess, generally of the large solitary type which occupies either right or left lobe of the liver, but generally the posterior aspect of the right (see Hepatic Abscess). The spleen, lungs, and kidneys may be enlarged and congested.

Symptoms.—The disease is often preceded by some disorder of the digestive tract, such as dyspepsia, with a coated tongue and irregularity of the bowels. Then follow rigors, with distinct fever, and a griping uneasiness in the belly. It is not, however, till the characteristic diarrhœa has occurred that the disease can be with certainty recognised. The first evacuation usually consists of the ordinary contents of the rectum, expelled it may be with more force than is usual ; then follow some scybala, often coated with mucus ; these are succeeded by frequent motions consisting (according to the stage of the disease) of thin mucus, blood, or a slimy material, highly offensive, of a pale yellow or buffy tint, and

often full of air bubbles, so that they resemble brewer's barm ('fermenting stool'). Later on, the motions may appear like jelly, or frog-spawn. They always contain some of the histological elements of the mucous coat of the large intestine which have been detached or altered by the inflammatory process, such as pus, blood, cylindrical epithelium, and necrosed patches of the mucous membrane of varying sizes and extent. The number of stools varies considerably; but they may amount, during the severest part of the attack, to as many as twenty an hour.

In addition to the diarrhœa there is a constant tenesmus, with a sense of pain and heat round the anus, or in the course of the colon, accompanied by tormina, tympanites and borborygmi.

The tongue is heavily coated; vomiting and belly cramps are frequent. There is constant micturition, although the quantity of urine is diminished, the diminution varying in degree according to the urgency of the diarrhœa and vomiting. The pulse is quickened; the skin is dry and hot. As the disease advances the features become pale and pinched, and the strength rapidly fails; but delirium, as a rule, is absent.

The disease may terminate: 1. In RECOVERY, when, after an illness varying from three days to a week or ten days, the evacuations become diminished, and contain more bile; tenesmus and other distressing symptoms are relieved; and the patient's strength gradually rallies. 2. In DEATH, preceded by exacerbation of symptoms; or by a remission following apparent improvement, when the patient has a quick running pulse, and gradually sinks from sheer exhaustion. Or 3. In CHRONIC DYSENTERY, in which the symptoms continue, but in diminished severity, with alternating exacerbations. The evacuations are generally loose, occasionally blood-stained, and contain mucus, pus, and endothelial sloughs, although occasionally healthy motions are passed. Tormina are a constant and distressing sign; abdominal tenderness is complained of, being often distinctly localised to certain areas of the large gut, which may be thickened to such a degree as to be easily felt on palpation. The tongue is abnormally red and glazed;

the appetite poor or capricious ; there is gradual loss of flesh ; the patient becomes anæmic ; whilst the mental condition is one of great irritability, or of indifference to recovery, or of hypochondriasis.

Diagnosis.—1. FROM PILES.—Internal hæmorrhoids may give rise to hæmorrhage and tenesmus. But the diagnosis would be cleared up at once on digital examination. 2. FROM STRICTURE AND ULCERATION OF THE RECTUM.—This condition is frequently syphilitic ; it occurs oftener in women ; it is not accompanied by any rise of temperature ; there is no great amount of mucous discharge. If any doubt exists, the rectum should be explored.

Prognosis is influenced greatly by the previous habits and the constitution of the individual. The mortality is increased in persons of depraved habits ; it is higher in the epidemic than in the sporadic form. Severe hæmorrhage is a grave symptom.

Treatment.—(a) PROPHYLACTIC.—Sanitary and hygienic rules are most important. Food should be thoroughly wholesome and sound. Drinking-water should be pure and fresh, and efficiently filtered ; if there is any probability of its contamination by dysenteric sewage it should be refused, or boiled previous to use. It were better to take the water-supply from a level higher than the area in which the disease is supposed to have originated. Other good precautions to be observed are, the avoidance of errors of diet, and of cold.

(b) CURATIVE.—The diet should consist almost entirely of milk, eggs, and farinaceous foods. Avoid animal food, especially salt meats. Effervescent wines, and beers are to be forbidden. Various medicines have their different advocates. A dose of castor oil at the onset may be given to clear the bowels of irritating contents. Then give ipecacuanha ; it has almost a specific action. The simple powder is the best, in twenty-grain doses every four hours. Such large doses are well tolerated. Vomiting may be controlled by small doses of opium. In Dover's powder the dose of opium is relatively too large. Warburg's tincture is largely prescribed in India, with good results. Tenesmus may be relieved by enemata of

starch and opium, or by leeches applied to the anus. Give hot fomentations to the abdomen, if there be much pain.

The treatment should be continued until all signs of tenesmus and mucous stools have disappeared.

In the Chronic form the chief object in treatment is to avoid errors of diet. The disease is soon rendered acute again by injudicious feeding; and it is astonishing how people who suffer from chronic dysentery frequently show an obstinate disregard to dietary rules.

Avoid all salt meats, highly peppered or seasoned dishes. The diet should consist mainly of milk, eggs, and starchy puddings, with an occasional allowance of fresh fish or underdone mutton. All animal food, however, should be forbidden on any exacerbation of symptoms. Intestinal irritation should be treated with alkalies (sodii bicarb., potas. bicarb., &c.), or with bismuth, or small doses of opium. Sulphate of copper (gr. $\frac{1}{2}$), the liquid extract of bael fruit (ʒj.), or the vegetable astringents (tannin, kino) may be used with advantage. Others have recommended large astringent enemata, such as sulphate of copper (gr. ij. ad ʒj.), or of nitrate of silver (gr. $\frac{1}{4}$ ad ʒj.). (See Stephen Mackenzie, 'Trans. Med. Soc.' 1882.)

Ipecacuanha in small repeated doses is beneficial in preventing the attack from again becoming acute. Warm clothing is indispensable; and a sea voyage should be ordered, where practicable.

LEPROSY (ELEPHANTIASIS GRÆCORUM)

Definition.—A specific disease, occurring endemically in certain parts of the globe, characterised by the development of inflammatory, nodular growths affecting the skin and nerves, followed by anæsthesia and death of the parts affected.

Etiology.—(a) **PREDISPOSING CAUSES.**—*Age.*—Adults most frequently affected. It is rare before puberty. *Sex* has no influence; men and women are equally liable. *Climate and Locality.*—It prevails, to any extent, only in tropical climates, but lingers in Norway and other parts of

Scandinavia. No visitation has occurred in Britain since the end of the eighteenth century. In those countries where the disease is endemic, it is found in marshy districts, on the banks of large rivers, and on the sea-coast. *Surroundings*.—It especially attacks the poorer population, amongst whom dirt, squalor, and unwholesome dietary are usual. Fish, as a constant food, has been suggested as a possible predisposing, if not the exciting, cause, owing to its presumably containing the specific bacillus ; but there is no confirmatory evidence.

(b) EXCITING CAUSE.—A specific micro-organism (*Bacillus Lepræ*) which, in its form and behaviour to staining reagents, strongly resembles *B. Tuberculosis*. The disease is not contagious, or only to a slight extent.

Pathology.—The disease appears to be essentially of a chronic, inflammatory nature, producing a proliferation of the cellular elements of the tissues involved. The organs, and especially the skin, are affected by an invasion of granulation tissue, in which small cells with large nuclei are prominent. The sheaths of the nerves become thickened, the inflammation attacking not only the perineurium, but the epineurium as well. The nerves also swell, and become moniliform. The muscles are affected with fatty degenerative and fibrous changes, and the bones atrophy. In the sloughs of the skin, and in the characteristic tumours, the specific *Bacillus Lepræ* (Heuser) has been found.

Symptoms.—The onset is gradual. The patient complains of debility, weariness, loss of appetite, and general malaise. Then patches of hyperæmia, red or copper-coloured, appear on the skin in different parts ; the skin over these areas is puffy and tender ; the redness tends to spread centrifugally, thus forming a central white blotch from which the ordinary pigment has disappeared, and leaving the sweat-pores open and patent, so that the skin bears some resemblance to the rind of an orange. Favourite sites for these outbreaks are the forehead, nose, ears, and the cutaneous distribution of the ulnar nerve. As these spots disappear, renewed outbreaks occur in other localities.

In another form, although the hyperæmic patches exist,

cutaneous anæsthesia is the most distinctive feature. This anæsthesia may appear in patches on different parts of the trunk, hands, and feet ; or it may be diffuse, especially in the head and neck. The skin over these areas is pale, glistening, and atrophied ; the hairs become grey, and fall out ; and there is a tendency to the formation of bullæ and ulcerations of the skin over the small joints of the hands and feet, with destruction and exfoliation of the bones. Spontaneous amputations of the phalanges may result from the ulcerations.

In a third variety, tubercular lumps form in the skin of the face, ears, and extensor surfaces of both extremities. The tumours are hard, and vary considerably in size and colour. They may be discrete or confluent, and when the face and ears are involved they produce great and often hideous deformity. They may disappear entirely, or leave their traces in the form of pigmented patches, destitute of hair, so as to resemble the scars of burns ; or, as the tumours increase in size, the skin over them may ulcerate, forming indolent offensive sores which extend deeply and cause destruction of bones, tendons, and joints in their neighbourhood.

Nor are these affections limited to the skin. The eyes also may become involved, a destructive form of conjunctivitis with sloughing of the globe being fairly frequent.

Similar growths may appear in the mouth and fauces, causing the tongue to become enlarged, nodulated, and fissured ; in the nasal cavities, with ulceration and destruction of spongy bones ; and in the larynx, which may be thickened and stenosed.

Exacerbations and remissions of fever accompany the increase and decrease of the tubercular growths.

All the above symptoms may occur in one individual, but varieties of leprosy (*L. Tuberosa*, *L. Anæsthetica*, *L. Nodosa*), have been described according as the different symptoms predominate ; but at best the subdivision appears artificial.

Prognosis.—Always unfavourable ; death occurring in from two to sixteen years. The termination of the disease is marked by gradual exhaustion, accompanied by dysentery, by phthisis, or by disease of the kidneys.

Treatment.—An incurable disease. Remove the patient from the locality where the disease is endemic. Attend to his general health. Allow a generous diet. Symptoms may be relieved by hot baths and the inunction of stimulating oils. Various balsams and oils have been administered internally and externally, but, so far, without marked success.

SYPHILIS

Definition.—A specific, eruptive disease, received only by direct inoculation ; characterised by a lesion at the point inoculated, with inflammation of neighbouring lymph-glands ; by subsequent lesions of the skin and mucous surfaces ; and, ultimately, by lesions of viscera, bones, muscles, and the deeper tissues. The inherited form of syphilis presents some modifications of the above. (See Congenital Syphilis.)

Causation.—(a) **PREDISPOSING.**—Although an almost universal disease affecting all nationalities, all ranks, and all ages, there are certain factors of predisposition, viz. : *Age.*—Young adolescents are more susceptible to the disease than elderly people. *Sex.*—In an equal number of exposures, women present a preponderance of cases, owing to the larger area of the mucous tract of the genitals, the most frequent site of inoculation. *Race.*—Orientals and dark races appear especially prone to contract the disease, and certainly, so far as one's experience goes, the type of disease is more virulent in dark races. *Station of Life.*—All ranks suffer ; but the disease appears to be more severe in the poor and ill-nourished.

(b) **EXCITING CAUSE.**—A specific poison received directly into the blood by some abraded surface, either during the sexual act (the great majority of cases), by suckling, by kissing, by exposure of hands during surgical operations on a syphilitic patient, or it may be introduced into the system by unclean surgical instruments (vaccination, circumcision).

An attack of syphilis, as a rule, gives perfect protection ; but second seizures are by no means unknown.

Incubative Period.—About thirty days. Hutchinson says it is nearer five weeks.

Symptoms.—The course of the disease has been divided into three stages : named PRIMARY, SECONDARY, and TERTIARY. This division is generally accepted, and is clinically convenient though somewhat arbitrary, as the stages are not always distinct, and often overlap each other.

1. PRIMARY STAGE.—After the period of incubation, there appears at the seat of inoculation a small papule of dusky colour, generally painless, with a tendency to enlarge slightly. It eventually sloughs at the summit, leaving, in about seven days, a small, circular, sharply-defined ulcer surrounded by an inflammatory, hardened, 'gristly' zone. This constitutes the hard or 'Hunterian' chancre. It is completely developed at about the sixth week, then begins to diminish, and ultimately heals, leaving a permanent scar. During the development of the sore the lymph-glands of the affected area enlarge painlessly, and become indurated like bullets. The induration may remain for some months.

2. SECONDARY STAGE.—Is characterised chiefly by slight fever and malaise, attended by cutaneous eruptions, by symmetrical ulcerations in the tonsils and fauces, and by inflammatory affections of the eyes and of the periosteum. The febrile disturbance, as a rule, is not excessive ; but is most marked during the evening. The cutaneous eruption is mostly found on the front of the chest or front of the arms, but may be present on any part of the surface. It is at first a mottled, 'measly' rash (*S. Roseola*), appearing in crops ; the spots subsequently becoming elevated and occasionally grouped in patches. As a rule, there is no itching.

Subsequently there ensues superficial, painless, and symmetrical ulceration on the tonsils, or pharynx, or soft palate ; or mucous tubercles form on the inner surface of the cheeks, on the tongue, at the angles of the mouth, the anus, vagina, or any soft mucous surface, with induration of the neighbouring lymphatic system. The hair falls in patches. Pains, due to slight transitory periostitis, supervene, especially at nights, in the tibiæ, clavicles, and the skull bones, and also in the joints. The secondary symptoms affecting the eyes are usually iritis

with irregular, and it may be fixed, pupils ; or, more rarely, general retinitis.

To revert to the cutaneous eruption (*syphilide*), it should be noted that it may be entirely absent ; that it often develops into scaly papules ; that it may vary in type from lichen to pustule ; indeed, that it may be imitative of any form of skin disease, and that different types of skin eruptions may present themselves in the same individual.

Hutchinson draws attention to an INTERMEDIATE STAGE, mainly characterised by psoriasis of the palms and soles, and by enlargement with stony induration of one or both testes (sarcocele), and by tenderness of liver and other organs.

3. TERTIARY STAGE.—The symptoms marking this epoch usually supervene about twelve months after contact, though they may occur before the second stage is over, and may continue for a number of years. Gummata due to hyperplasia of tissue elements, thus forming distinct tumours, are the most characteristic phenomena (see p. 40). Gummata tend to spread ; they are often attended by recrudescence of fever ; and after their subsidence leave permanent destruction of the parts affected, or hardened, atrophied cicatrices. They may involve the bones, causing chronic, diffuse periostitis, nodes, or necrosis ; they may form in the voluntary muscles, in the viscera, in the connective tissues and other fibrous structures, in the skin, forming serpiginous ulcerations, or in the tongue, leading to sclerosis of that organ. The various joints of the body, especially the larger ones, become the seat of chronic inflammatory changes. Indeed, no tissue or organ of the body appears to be exempt from attack. Beyond these affections there may be chronic changes in the nervous system, either in the form of localised tumours, or of chronic inflammatory changes (sclerosis) in the brain or cord, and nerves issuing therefrom. Thus, certain nervous tracts are involved in the brain, and subsequently in the spinal cord ; or the changes may originate in the cord and its membranes ; and in any case they are frequently notable for their irregular and disseminated distribution. The various mucous tracts are also prone to pathological changes. They

tend to become thickened, owing to low inflammatory processes ; and ultimately to ulcerations, leading to strictures and stenoses of canals and ducts. Thus, as regards the digestive tract, we find destructive ulcerations or narrowings in the fauces, pharynx, and œsophagus, and a like condition in the rectum, especially in women. Similarly, deep ulcerations may occur with destruction of cartilaginous structures in the larynx and trachea, followed by stenosis.

Subsequently, in chronic cases, amyloid degenerative changes supervene, affecting principally the liver, spleen, kidneys, and intestines, accompanied by marked cachexia, and increase of white corpuscles in the blood. These changes frequently induce general tuberculosis or lymphadenoma as a termination of the disease.

The above is a brief sketch of the signs common to the tertiary stage ; but a further enumeration of changes which may occur in the various systems is advisable. It will be noted, however, that it is impossible to confine an account of the various lesions to individual systems ; for example, the affections of the spinal cord frequently commence in the blood-vessels in the first instance.

(a) NERVOUS SYSTEM.—Perhaps the most important of the tertiary lesions which come under the observation of the physician are those of the nervous system. 1. *Meningitis* is a frequent occurrence, which may or may not be followed by general paralysis, mania, dementia, or other abnormal mental states, especially in such cases as are complicated by anxiety or overwork. 2. *Paraplegia* may occur, either dependent on gross lesion of the cord followed by softening ; or on the mechanical effect of a localised gumma ; or on thrombosis or other derangement of the vascular supply of the cord. Recovery of function takes place, as a rule. 3. *Ophthalmoplegia interna*, the pupil being fixed with no accommodation to light. *Ophthalmoplegia externa* with paralysis of muscles supplied by the third, fourth, and sixth cranial nerves. 4. *As a Sequela*.—In neglected cases, ascending spinal paralysis or locomotor ataxy may supervene. As regards locomotor ataxy, however, it is probable that sexual excesses and muscular

fatigue are most important factors. Syphilis alone will scarcely account for the great number of ataxic cases (80 per cent., Erb), as this neurosis is rare in women who have had syphilis.

(b) VASCULAR SYSTEM.—Obliterative arteritis, especially of the cerebral vessels ; cerebral thromboses ; chronic inflammatory and ulcerative changes in the aorta or large arteries, leading to aneurysm.

(c) SPECIAL ORGANS.—*Eye*.—Acute iritis, or scattered patches of choroiditis are most common. *Ear*.—Chronic inflammatory changes in the middle and internal ear, causing permanent deafness.

(d) VISCERA.—*Liver*.—The disease gives rise to a coarse and insular form of cirrhosis ; or the formation of gummata with jaundice, due to mechanical obstruction of the bile ducts. *Spleen*.—This organ may be enlarged from a general increase of its histological elements ; or it may be the seat of gummata. *Lungs* may be implicated with chronic, inflammatory changes (fibroid phthisis), or with scattered gummata. *Heart*.—Similar changes may occur in the muscular walls, or in the various valves. *Testes and Ovaries* not infrequently bear evidence, by their chronic, hardened, and enlarged condition, to the existence of previous syphilitic attack.

Treatment.—Mercury in one form or other, either by the mouth, or by inunction, or by subcutaneous injection, or by fumigation, has now become the almost universal remedy. Certainly no sufferer can be said to be cured who has not, at one time or other, undergone a course of the drug.

Iodide of potassium is required in the later stages, or after mercury appears to have had its effect. It often appears to awaken the action of mercury.

For the PRIMARY SORE little is required beyond cleanliness and protection. The parts should be thoroughly washed ; then apply a mercurial lotion (black wash : calomel gr. x., aquæ calcis ℥j.), or dust the sore with iodoform, or apply iodoform ointment (1 in 8). Cauterisation or excision of the sore appears useless, although advocated by some high authorities. But mercury should be administered immediately the diagnosis

of syphilis is certain. Give gr. iij. of the blue pill, or gr. iv. of the grey powder, or the green iodide (gr. j. to gr. ij.), three times a day. Any tendency to diarrhœa may be checked by adding small doses of opium. This treatment should be continued for six weeks or two months ; but it is not desirable to produce salivation.

In the SECONDARY STAGE the eruption may be absent, especially if mercury has been given from the onset of the disease. In hospital practice it is probable that most cases present themselves at this period. Mercury, however, should still be administered, unless there be serious contra-indications. It is best given by the mouth ; inunction, fumigation, and hypodermic injection being kept as reserves. If inunction be desirable, begin with ʒj. of the blue ointment, which may be rubbed into the inner aspect of the thighs, or into the skin of the chest and belly, at bedtime ; the greasiness can be easily removed in the morning by warm soap-and-water. Fumigation may be used when a rapid action of mercury is necessary. Calomel (gr. xx. to gr. xxx.) or the bisulphuret of mercury (ʒj. to ʒij.) are the best preparations. Use a Lee's lamp by which the body is steamed before the mercurial is volatilised. Hypodermic injections may be performed once a week into the buttock. Gr. $\frac{1}{3}$ of the bichloride in ℥xxx. of water is recommended by Mr. Bloxam. If *iritis* is present, secure dilatation of the pupil by the application of a solution of atropine to the conjunctivæ (gr. v. to ʒj.), but continue the mercury. The patient should rest in bed, in a darkened room, and he should live abstemiously. *Periostitis*, *Alopacia*, and the skin affections all require the same general treatment. Iodide of potassium, though prescribed by some medical men, does not appear to be so beneficial.

In the TERTIARY STAGE, iodide of potassium may be combined with mercury with advantage. Give potassium iodide (gr. v.) with liq. hydrarg. perchlor. (℥xxx.) in some bitter infusion three times a day. Mercury seems to be of especial service in arteritis, iritis, and in disease of the nervous tissues, especially when given in prolonged small doses ; on the other hand diseases of bones and lupoid affections of skin

appear to yield quicker to iodide of potassium by the mouth, or as an ointment. Both together give rapid results in cases which are at all amenable to treatment. But it will often be found that small doses of iodide produce better results than the larger ones. In cases of heroic doses (gr. xx.) the patient not only becomes depressed, but much of the drug passes off immediately by the urine.

The results of treatment during the tertiary stage, however, are often variable and uncertain. Relapses are common after leaving off treatment. According to Gowers, syphilitic lesions of the nervous system are seldom or never cured ; a statement which, notwithstanding the eminence of the authority, appears doubtful.

Rules for the Administration of Mercury.—1. It is not so much the quantity that is wanted, as its effect on the system ; therefore, individual susceptibility must be considered. 2. Mercury can always be tolerated if the proper dose be found. 3. It should be given to a syphilitic patient who is about to marry, right up to the date of marriage ; but wedlock should be forbidden until two years at least from the date of onset of the disease. 4. During the administration of the drug the patient should be forbidden to smoke, or to take coffee, fruit, and aperient medicines ; the teeth, gums, and buccal cavities should be carefully washed and cleansed. 5. The drug should be watched, and given cautiously to patients with Bright's disease, and to those suffering from marked anæmia or from strumous diathesis. 6. In a case of syphilitic pregnancy give the mother small doses until parturition.

Note that syphilis tends to spontaneous cure in many cases, or at least, as time progresses, to diminish in activity ; that, as in all specific diseases, it affects different individuals with varying degrees of virulence : in some it is a malady seriously threatening life, in others it is an extremely mild disorder. Its operations are different in various races, being particularly severe amongst the inhabitants of India and of other tropical countries.¹

¹ See Hutchinson's *Syphilis*.

CONGENITAL SYPHILIS

Definition.—Syphilis acquired from one or both parents *before birth*.

Symptoms.—The typical manifestations usually appear about the sixth week, a child born with a syphilitic eruption being exceedingly rare. There is no order of symptoms, except that the child comes into the world with ‘secondaries,’ or develops them shortly afterwards. The supervention of snuffles, with a peeling erythema of face and nates, are usually the first signs. The bridge of the nose is widened and sunken ; there are tender areas on the skull, especially in the parietal and frontal bones ; the epiphyses of the long bones of limbs are also tender, and sometimes swollen. The skin, especially of the face, is soft, thin, and pallid, but often ‘muddy ;’ and the child, generally speaking, is puny and badly developed. After twelve months, unless previously treated, other symptoms now appear, whilst the snuffles and rash have vanished. A permanent physiognomy is usual. The head is enlarged ; the parietal and frontal prominences are more pronounced ; the angles of the mouth are fissured and puckered ; the irides assume a greyish tint ; the corneæ are duller than in health ; and there is a frowning expression due to photophobia. The nose is still sunken and flattened, and the palpebral fissure is narrowed, so that the child acquires a somewhat Chinese type of face. Characteristic changes occur in the teeth, both temporary and permanent ; they are ill-developed, narrowed, and fissured, eventually showing a well-marked notch like a ‘rifle-sight’ in their free edges. Should these appearances occur in the central incisors, the evidence is almost conclusive. This condition should not be confounded with that due to stomatitis from prolonged use of mercury, when the teeth are decidedly peg-shaped. Some authorities, however, look upon a peg-shaped tooth as being suspicious of syphilis.

Other affections are principally those involving the nervous system, and the organs of special senses. Any lesion or condition of the nervous system may occur, such as epilepsy, arrested development of brain with idiocy, deaf mutism,

ophthalmoplegia externa ; but locomotor ataxy and Bell's palsy are said to be rare. As regards the *ear*, a child presenting otorrhœa after the twelfth month, with subsequent deafness, would certainly be very suspicious of syphilis. Similarly in the *eye*, a symmetrical keratitis, with signs of ulceration in the soft palate, renders a diagnosis of syphilis almost certain. Iritis and choroiditis may appear at about the fifth month, especially in those children begotten at an early period of the parental disease. The innermost tunic of the eye may also be affected (neuro-retinitis), often in patches.

In addition, we must remember that there is, as in acquired syphilis, a tendency to inflammatory changes (gummata) in all the viscera, connective tissues, and bones ; that the hair and nails tend to defective growth, or to fall off ; and that the development of the body generally is often stunted, and that of the mental faculties arrested.

In cases of doubt as to symptoms we must inquire into the parental histories, especially the mother's. Miscarriages in sequence are generally due to syphilis, but not always ; similarly, a dead and decomposing foetus is very often a result of syphilis, but it is not by any means conclusive.

Prognosis.—Unfavourable in poor children, owing to their unhealthy surroundings. On the other hand, the children of the rich tend to recovery under careful treatment. Relapses are rare. It is also uncommon to find the disease transmitted to a third generation ; yet such cases have been recorded.

Treatment.—Give mercury as soon as the diagnosis is confirmed. Children bear the drug well ; but do not prolong it more than is necessary, owing to its effect on the teeth. It may be given by the mouth. P. hydrarg. c. creta (gr. j.), thrice daily, acts very efficiently, and can be easily administered. Sometimes very speedy results are obtained by inunction. Rub gr. x. of the ungt. hydrarg. into the soles and palms night and morning. Iodide of potassium is too depressing. Still, it may be given in small doses (gr. j.) after a course of mercury ; it then appears to increase the activity of the latter remedy. Ulcers and painful excoriations of the skin are also best treated by mercurial ointment, or by the application of

calomel. The erythema on buttocks and nates will be considerably relieved by dusting with a compound iodide of lead powder (Plumbi Iod: $\bar{5}$ jss.; Plumbi Carb: $\bar{5}$ vj.; Pulv: Amyli $\bar{5}$ ijss. Misce).

Beyond all this, it is necessary that the child's general health should be attended to. The infant should be removed from all unhealthy surroundings, and carefully fed on good, sound food; it should be warmly clothed, and have an abundance of light and fresh air. The mother of a syphilitic child may suckle her own offspring and not be infected, but not so a wet-nurse.

GLANDERS (EQUINIA)

Definition.—A specific disease affecting horses and mules, whence it is communicated to man, in whom it is characterised by an inflammatory, nodular affection of the mucous membrane of the nose and air-passages, and, secondarily, of the skin, lymphatic glands, and other organs, together with the subsequent formation of abscesses.

Causation.—(a) PREDISPOSING CAUSES.—*Occupation.*—It is found amongst ostlers, grooms, and those brought in contact with horses.

(b) EXCITING CAUSE.—A specific bacillus somewhat resembling the *B. tuberculosis*. The disease appears to be special to horses and kindred animals, just as rabies is special to dogs. For this reason it would seem to be particularly fatal when introduced into the human system. (See Specific Fevers, p. 98.)

Incubative Period.—Not accurately ascertained. It varies from one to fifteen days; a week being probably near the length of incubation.

Pathology.—The disease is marked by the formation of tuberculated new growths in the skin, mucous passages, and organs of the body. Like similar formations in true tubercle, they tend to undergo inflammatory and retrogressive changes terminating in suppuration. The muscles are soft; they are frequently the seat of hæmorrhages, which subsequently suppurate. In the lungs we find bronchitis, patchy pneumonia, or pleurisy.

Symptoms.—(a) PRIMARY FEVER is characterised by rigors, sweats, headache, constipation, and marked pyrexia; followed by diarrhœa. The duration of this stage is uncertain.

(b) ERUPTIVE PERIOD.—The mucous membrane of the nose and naso-pharynx is congested, discharging a watery, acrid fluid, which subsequently becomes tenacious and finally replaced by sanious pus. Subsequently in this mucous tract small pimples form, hard and shotty in character, reaching the size of peas. They afterwards pass through the stages of vesicle, and pustule, and finally slough, leaving punched-out ulcers with red, elevated, erysipelatous margins. During the pustular stage the eruption may be confluent, as in small-pox. At the same time there may be a purulent form of conjunctivitis; sores may appear in the buccal cavities, causing great fœtor of breath; or there is an erysipelatoid affection of the nose, cheeks, and eyelids, together with inflammatory extensions to the bronchi, lungs, and pleuræ. Abscesses tend to form in various parts of the body, especially in the neighbourhood of joints. Fever continues high (104° to 105°), and is accompanied by diarrhœa, with fetid stools, great prostration, and a tendency to the 'typhoid' condition, which terminates in delirium and coma. Death usually supervenes in the second week, although in some cases the illness drags a slow chronic course. It will be observed that in some points the disease bears a resemblance to small-pox and to pyæmia; and, as in small-pox, its severity is increased when acquired by inhalation as compared with inoculation. Further, the disease varies somewhat in its symptoms when inoculated on some abraded surface. Here the signs are most pronounced in the skin and lymphatic glands. At the point of inoculation a sore rapidly forms, with hardened, tender, and dusky red areola. The sore rapidly ulcerates, and tends to spread, forming an unhealthy chancre exuding offensive sanious pus. The neighbouring lymph vessels and glands become cord-like, knotty, and indurated ('farcy buds'); there are severe fever and subsequent deep-seated abscesses; but the nasal symptoms are not so prominent, nor is the eruption on the skin and mucous

surfaces so pronounced, as in that form of the disease which is received by the respiratory tract.

Diagnosis. FROM PYÆMIA.—The early history of the disease will reveal the presence of the characteristic eruption of glanders. In the later stages, when abscesses have formed, the diagnosis is more difficult. Inquiry should be made as to possible contact with diseased horses.

FROM ACUTE RHEUMATISM.—Here the absence of eruption, the profuse, sour sweat, the metastatic character of the arthritis, should form a sufficient guide.

FROM TYPHOID FEVER.—Examine for rose rash, also for characteristic diarrhoea, and other abdominal symptoms. The temperature curve is less markedly intermittent than in equinia.

Prognosis.—Always grave. Recovery may take place in chronic cases, and in those in which the poison has been received by direct inoculation.

Treatment.—There is no specific remedy. Treat the case as in pyæmia. Cleanse the mouth and buccal cavities (Condy's fluid, carbolic acid), and open superficial abscesses with antiseptic precautions. Be careful to avoid self-inoculation when dressing the lesions. Quinine, arsenic, and other tonics should be given, together with a stimulating diet (alcohol, eggs, beef-tea).

ANTHRAX (MALIGNANT PUSTULE, WOOLSORTERS' DISEASE)

Definition.—An acute, infectious, and contagious disease, received by the air-passages, or by the digestive tract, or by direct inoculation; characterised by fever, catarrhal affections of mucous membranes, and a typical lesion at the point inoculated.

Causation.—(a) PREDISPOSING.—*Occupations.*—It is especially liable to prevail amongst those who are in contact with herbivorous animals or with their skins. Hence, it is most frequent in butchers, flockmasters, shepherds, tanners, and also in the woolsorters of Bradford and elsewhere.

(b) EXCITING CAUSE.—A specific micro-organism—the

Bacillus anthracis. This bacillus is rod-shaped, tends to grow quickly, but breaks up eventually into cylinders about $\frac{1}{2000}$ of an inch in length, containing spores.

The bacillus, if acquired by inoculation, produces a typical lesion at the point of contact. If it is received by the stomach, by means of contaminated milk, for example, or by the respiratory tract, the symptoms are entirely constitutional.

Symptoms may, therefore, be divided into two categories : (a) LOCAL or EXTERNAL ANTHRAX, and (b) CONSTITUTIONAL or INTERNAL ANTHRAX ; the disease thus bearing comparison with farcy and glanders.

(a) LOCAL or EXTERNAL ANTHRAX, or MALIGNANT PUSTULE.—At the point of inoculation there forms, in a few hours, a papule which ultimately develops into a vesicle. This is surrounded by a zone of highly-inflamed skin and subcutaneous areolar tissue. Eventually the vesicle sloughs, leaving a crateriform sore with a brown, unhealthy-looking sphacelus on its floor. From this centre, lymphatic inflammation extends upwards, and produces a tense, brawny infiltration of the limb or part affected, together with inflammation and induration of the neighbouring lymphatic glands. All these conditions are accompanied by fever, which may not, however, be severe until the sloughing and surrounding induration are well marked. In three or four days more—*i.e.* about the sixth or seventh from inoculation—there is, if the case terminates favourably, a marked fall of temperature, the eschar separates, and recovery is gradual. On the other hand, diarrhœa with prostration, delirium and increase of fever may ensue ; these symptoms being usually fatal.

In another case there would appear to be no signs of a local infection ; but a severe œdema ensues, limited to the face, the eyelids, or to a limb. This œdema may go on to gangrene, with marked fever, delirium, and the typhoid state, which precede death.

(b) CONSTITUTIONAL or INTERNAL ANTHRAX is produced by eating the flesh, or consuming the milk, of animals which have been infected with the disease.

The symptoms in one series of cases are such as would be

produced by an irritant poison affecting the gastro-intestinal canal. There are, therefore, chills, rigors, and general pains, with severe diarrhœa and vomiting; the spleen is enlarged; and occasionally a petechial eruption is seen on the skin, or hæmorrhage occurs from the mouth or rectum.

The symptoms in another series, again, are accentuated in the respiratory tract, the bacillus being breathed in with dust when sorting wool. The onset here is sudden. The patient complains of rigors, pains in the back, and vomiting. His respiration is rapid, the pulse is accelerated, fever is marked (temperature 103°), and on auscultation he presents the physical signs of acute general bronchitis. Death often occurs in forty-eight hours, with signs of collapse. If life be prolonged over this period, the fatal event is preceded by diarrhœa, with delirium or unconsciousness.

Diagnosis.—When the characteristic pustule is seen, the diagnosis is easy. In internal anthrax the diagnosis is more difficult. We must suspect the disease from the acuteness and severity of the bronchial or intestinal symptoms, if they occur in a person employed amongst raw, unprepared wool or hides. The cultivation of the bacillus would be conclusive.

Prognosis.—The disease is not necessarily fatal if recognised early and vigorously treated; but it is extremely fatal in neglected cases.

Treatment.—(a) **LOCAL.**—The point of inoculation should be excised as soon as possible, or destroyed either by caustic or by the hot iron. Prevent the spread of the bacillus by a girdle of subcutaneous injections of carbolic acid (2 per cent.) or of bichloride of mercury ($\frac{1}{10}$ per cent.), and repeat these procedures two or three times a day.

(b) **INTERNAL.**—A purgative may do good in cleansing the bowels of the offending organism. After that, all that we can do is to treat symptoms as they arise, and to maintain the patient's strength by an abundance of good food, together with alcoholic stimulants. Mr. Davies-Colley gives ipecacuanha (gr. v. to x. of the powder every four hours) with success. He also applies the drug directly to the wound after excision.

HYDROPHOBIA

Definition.—A specific disease, specially affecting dog, wolf, cat, and some other animals, which is imparted to man by inoculation only, and characterised by spasms of the throat, which are induced by attempts to swallow water.

Causation.—A specific virus which emanates from the salivary glands and buccal mucous membranes of a rabid animal. According to all bacteriological laws, the disease should never originate *de novo* ; and in some countries it is unknown, apparently owing to the rigid exclusion of dogs from other lands. Some veterinarians, however, tend to the belief that the bite of an infuriated dog, although the animal may be to all appearances perfectly healthy, may produce rabies in the bitten dog or man.

Men suffer more frequently than women ; they are brought more in contact with dogs. Only a certain proportion, variously estimated, however, of those who are bitten by a rabid animal contract the disease.

A bite on the hand, or face, or any part which is unprotected, is more likely to impart the virus than if it occur through the clothes.

Incubative Period.—This is very uncertain. It appears to vary from six weeks to twelve months, or even longer.

Pathology.—No definite post-mortem appearances have been found beyond congestions of the central nervous system, with slight invasion of leucocytes into the tissues. Vesicles have been described as occurring near the frænum linguæ some time between the third and the twentieth day after the bite.

Symptoms are divided into two stages, the first period being marked by depression and the second by excitement.

The stage of **DEPRESSION** is characterised by a feeling of malaise, or 'out of sorts.' The patient is anxious, restless, and sleepless, as he now vividly remembers the bite. Subsequently he may suffer from dryness or stiffness of the throat. The scar of the bite tingles or becomes painful, and there may be neuralgia

of the trunk nerves of the limb or part which is bitten. This pain of scar, in some cases, precedes all other symptoms.

The stage of **EXCITEMENT** then supervenes. The patient is terribly anxious ; his appearance is one of great terror ; he has delusions and hallucinations ; he is incoherent in his talk, but can easily be brought to his senses, and for a time may converse rationally ; but he is often petulant, and at such periods he may upbraid his doctor for leaving him. Then ensue hyperæsthesiæ of the skin and organs of special sense, any stimulation of which produces clonic and tonic spasms of various muscles, especially those of deglutition and respiration. Thus the various senses are irritable to light, sound, touch, and movements. He has spasms of the throat, which are excited by the sight or sound of water. He has thirst, yet cannot drink. He cannot even swallow his own saliva, which collects in the mouth and is worked up into a frothy condition. A noise, or a wave of cold air, will induce spasm of the respiratory muscles. In the interval, his breathing is sighing, or with effort. He has a hoarse cough or bark.

As the disease progresses, the spasms become more frequent and more prolonged. The patient gets weaker, and he dies generally about the third day from the onset of symptoms. The immediate cause of death is asphyxia after an attack of tetanic spasm of the respiratory muscles, or exhaustion.

Treatment.—(a) **PROPHYLACTIC.**—Immediately tie a ligature round the limb, above the point of inoculation. Cleanse the wound, and quickly apply the actual cautery, or excise it. There is no evidence to show how far these measures are preventive, but they are preferable to doing nothing.

(b) **CURATIVE.**—When the symptoms are established medicinal treatment is hopeless. Chloral hydrate, cannabis indica, and curare have all been advocated. Curare is the only drug which has the credit of effecting a cure.

The recent inoculation treatment of Pasteur appears to afford more than good hope of cure. The virus of rabies is passed through a successive series of rabbits until the incubative period is shortened to a week. A portion of spinal cord from the last animal inoculated is, after exposure to the air, which

lessens its virulence, used as a subcutaneous injection on the person who has been bitten, and after a series of such injections he is thus protected.

No one who has read Pasteur's and Horsley's papers on this subject can doubt the efficacy of this great discovery.

PYÆMIA

Definition.—A specific disease, characterised by fever of a remittent type and the formation of multiple abscesses, which are secondary to some previous collection of pus or to some open wound. The disease may run an acute or a chronic course.

Causation.—(a) **PREDISPOSING CAUSES.**—A wound or injury, or ulceration. An ill-ventilated and improperly cleansed ward, tent, or other apartment overcrowded by patients suffering from ill-dressed wounds. Drunken or debilitated constitution. Old age. Exhaustion and fatigue.

The wound itself, besides due to amputation or other major operation, may consist of an ulcer (of bowel), an abscess (of middle ear, or of prostate), an open sore (superficial burn or scald). The uterus after parturition would come under the same category as amputation.

(b) **EXCITING CAUSE.**—A specific virus, probably the *Streptococcus pyogenes*, as this is always found. This micro-organism circulates in the blood, and possibly sets up the febrile condition ; or it causes thrombosis at various centres, from which infective emboli are detached, which, again, originate the multiple infarctions and abscesses.

Cases are said to occur spontaneously ; but there is not sufficient proof, and such opinion is quite opposed to the modern pathology of micro-organisms.

Pathology.—The wound, or starting-point of the disease, is unhealthy-looking, foul, and offensive. If the lesion be superficial, its edges are swollen, red, and inflamed. The neighbouring lymphatic vessels are red and indurated, and the glands immediately above inflame and generally suppurate. Secondary metastatic abscesses are found in different tissues

and organs throughout the body. The lungs, pleura, or pericardium are most frequently involved in acute cases. In the more chronic forms there does not appear to be any regular order or preference beyond that that organ which is in direct communication with the irritant lesion is first affected. Thus abscess of the liver occurs when ulceration of the bowel is the primary cause of pyæmia; the kidneys are involved secondarily to bladder, prostatic, or urethral injuries; and splenic and other infective embolic infarcts originate from ulcerative endocarditis. Exceptions, however, to this rule must be made in the case of suppuration of joints and subcutaneous tissues. They appear to form more frequently in chronic pyæmia, although by no means unknown in the acute form of the disease.

Symptoms.—**ACUTE PYÆMIA.**—The onset of the disease is sudden. It usually begins with a severe rigor, or a series of rigors, each one of which somewhat resembles the cold stage of ague. This is succeeded by high fever (105° – 107°), a rapid pulse, quick respiration. The tongue is dry, there is great thirst, and often diarrhœa or vomiting. The patient's complexion is sallow or jaundiced, and his skin is bathed in profuse sweats. A peculiar sweet odour is generally exhaled from the skin and breath. He sooner or later develops in his lungs the physical signs of pneumonia or pleurisy. Pericarditis also may supervene rapidly; whilst in another case symptoms of meningeal inflammation may be revealed.

The wound from which the infection originates has an unhealthy appearance, with inflammation of the neighbouring lymphatic vessels and glands.

Death usually occurs in from five to seven days, being preceded by symptoms of severe exhaustion and by delirium.

It will be seen from the above that the symptoms are such as would be presented by the action of a virulent blood poison. The type of fever is markedly remittent: three or four extensive remissions may occur in the twenty-four hours; but there is no complete intermission, except in rare instances. The periods between the rigors and febrile exacerbations are short,

and produce not only bodily exhaustion, but great mental distress. The lungs, pleuræ, and pericardium are always secondarily involved.

IN CHRONIC PYÆMIA the symptoms are much the same in their sequence, but are less severe, and the periods between the rigors and febrile exacerbations are longer. There is also a tendency for the metastatic inflammation to miss the lungs and pericardium and to involve the larger joints, such as knees, elbows, and shoulders, and also the subcutaneous fascia and muscles. Hence, should the patient survive, it is at the expense of an ankylosed joint or joints, and probably of some scarring or disfigurement from the multiple abscesses. Death, on the other hand, may supervene from exhaustion after a struggle of some weeks' or even months' duration.

Diagnosis.—FROM AGUE.—In this disease, with which acute pyæmia may be confounded, the rigors are periodic, usually occurring at about the same hour each day, or each alternate day. The perspirations and prostration are not so intense, and the disease yields rapidly to the effects of large doses of quinine.

FROM ENTERIC FEVER.—Chronic pyæmia may bear some resemblance to this disease. In enteric fever, however, there is no marked rigor or series of rigors, the temperature is not so markedly remittent, and the presence of rose spots would render a diagnosis easy.

FROM ACUTE RHEUMATISM.—Here the arthritis is seldom so intense as to produce suppuration; nor does it linger in one joint, as in pyæmia.

In any case, the presence of an open wound in an unhealthy state, which is complicated by the onset of a rigor, with wide remissions of temperature, should always lead us to suspect pyæmic invasion.

Prognosis.—The acute form of pyæmia is generally a fatal disease, but not always so. Cases recover which are placed in advantageous surroundings of pure fresh air and good nursing. In chronic pyæmia the prognosis is more hopeful, especially if no vital organ be secondarily involved. In all cases much will depend on the constitution and vigour of the patient.

Treatment.—Remove the patient, if possible, from the infected room, and allow a maximum of fresh air. Give nourishing food, and plenty of it, such as beef-tea, eggs, milk, with alcoholic stimulants. Treat any superficial wound with the most rigorous antiseptic dressings.

Quinine is considered to be the only drug which has any controlling action on the poison. Give large doses (gr. x. every four hours). Hyposulphite of soda (gr. xx.), sulphocarbolate of soda (gr. x.), and salicylic acid (gr. x.) have also been recommended. Morphia may be required to produce sleep.

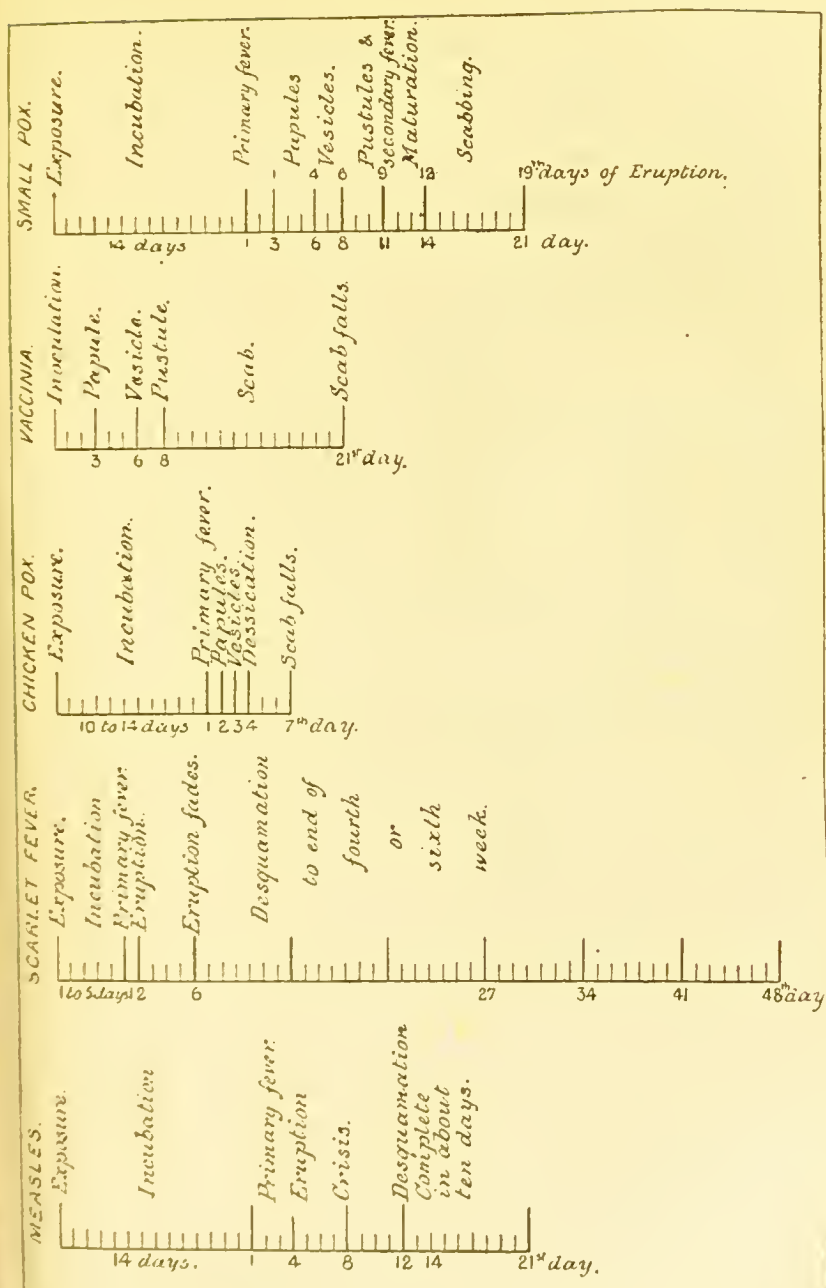


FIG. 7.—DIAGRAM SHOWING THE COURSE AND DURATION OF SPECIFIC FEVERS

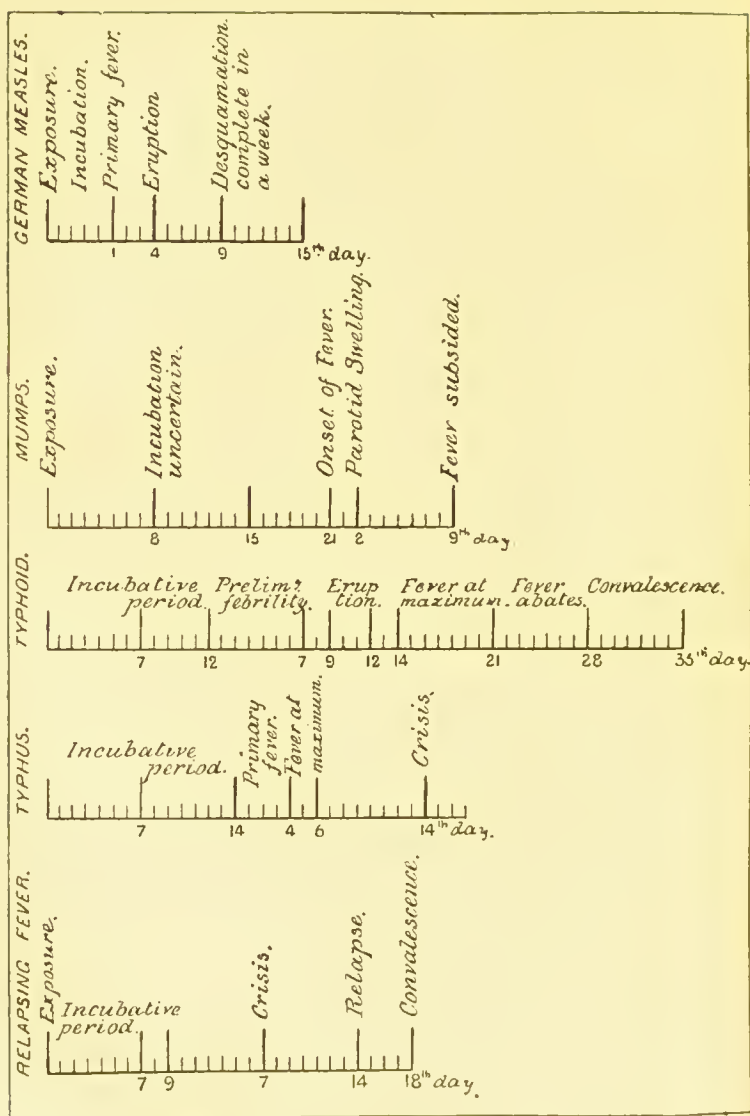


FIG. 8.—DIAGRAM SHOWING THE COURSE AND DURATION OF SPECIFIC FEVERS

DISEASES OF THE DIGESTIVE ORGANS

ANATOMY

The Mouth.—Two cavities are generally spoken of : an inner one, internal to the line of the teeth and gums ; and an external one, between the teeth and the cheeks.

The roof of the mouth is formed by the hard and soft palates ; the floor is formed by the tongue ; whilst the lateral boundaries are the teeth and gums or the cheeks.

The muscular reflex of the soft palate is extremely sensitive. The levator palati and azygos uvulæ are supplied by the small petrosal nerve from Meckel's ganglion. The tensor palati is supplied by a branch from the otic ganglion.

The Teeth.—In each lateral half of the jaws there are five teeth in the child and eight in the adult.

The first milk tooth (central incisor) appears about the seventh month, being contemporary with the commencing activity of the salivary glands. The last milk tooth (second molar) is cut at about the end of the second year. On account of the first permanent molars appearing before all the temporary teeth have been shed, a child's jaws at about $5\frac{1}{2}$ years may cause some complexity, as at that period the child would possess, besides the twenty temporary teeth, four permanent molars as well. As a rule, a tooth erupts in the lower jaw some few weeks before its companion in the upper maxilla. The following diagram (p. 196) shows the number of temporary and permanent teeth in each half of a jaw, with dates of eruption.

The Tonsils, one on each side, are situated between the anterior and posterior pillars of the fauces, palato-glossus and palato-pharyngeus, respectively. Each gland has about twelve

crypts ; it lies against the superior constrictor of the pharynx, but does not emerge from the pillars of the fauces towards the middle line of the mouth, nor does it extend outwards to the internal carotid artery, except the gland be enlarged. It has a rich vascular supply from the ascending pharyngeal, the facial, and the lingual arteries.

The **Œsophagus**, or **Gullet**, is a muscular tube, nine to ten inches in length, extending from the cricoid cartilage, opposite

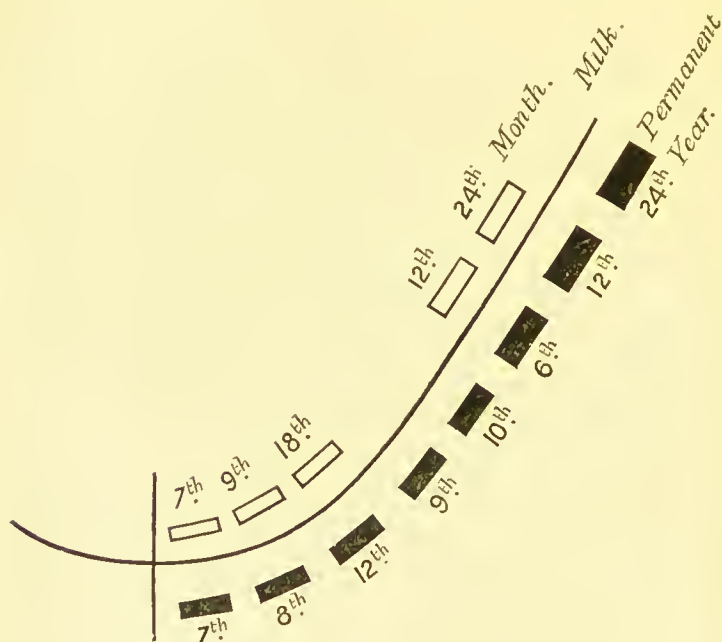


FIG. 9.—TO SHOW DATES OF ERUPTION OF TEETH

the sixth cervical vertebra, to the aperture in the middle of the diaphragm, opposite the ninth dorsal vertebra. It has thus a cervical and a thoracic portion.

In the neck it lies immediately behind the trachea, to which it is intimately attached, with an inclination to the left side. The lateral lobes of the thyroid body may also slightly overlap it. Behind, it is separated from the spinal column and the prevertebral muscles by a loose layer of areolar tissue.

On either side, lying in the groove between the œsophagus and the trachea, is found the recurrent laryngeal nerve ; whilst more externally it is in relation with the sheath of the large vessels, containing common carotid artery, internal jugular vein, and pneumogastric nerve.

The situation of stricture of the œsophagus is measured from the incisor teeth. The commencement of the gullet is six inches, and the cardiac sphincter of the stomach is about fifteen inches from these teeth. In the thorax it still keeps its posterior relation to the trachea, at the bifurcation of which it is placed somewhat to the left, so as to be behind the left bronchus, with the thoracic duct on its left side, and the vena azygos major vein on the right ; thence it descends behind the heart and pericardium, the lower part of the thoracic aorta being on the left of it, at about the fourth and fifth dorsal vertebrae, but entirely posterior to it, afterwards. The œsophagus is surrounded by a plexus of nerves chiefly from the vagus, the left nerve being for the most part on its anterior surface, the right nerve behind.

The **Stomach** is situated in the epigastric, the left hypochondriac, and partly in the right hypochondriac regions. Its size, and therefore its situation, will, however, vary considerably, according as the organ is distended or contracted.

Its anterior surface, which also looks slightly upwards, is in relation with the anterior wall of the abdomen in the epigastric region, and also with the under surface of the liver, and with the diaphragm.

Its posterior surface, looking backwards and downwards, is in contact with the pancreas, the third portion of the duodenum, the aorta with its cœliac axis and mesenteric branches, the inferior cava, the crura of the diaphragm, and the solar plexus.

The upper border is held to the transverse fissure of the liver by the lesser or gastro-hepatic omentum, which has between its folds the gastric and the pyloric vessels ; this border is also attached to the diaphragm by the gastro-phrenic fold of peritoneum. The lower border gives attachment to the great omentum, which contains the transverse colon.

This border is traversed by the right and left gastro-celiac vessels.

The pyloric end, more movable than the œsophageal, is in relation with the neck of the gall bladder and the under surface of the liver. The pyloric opening itself is not a valve, it is more or less a sphincter ; and in health, only admits the passage of one's little finger.

The splenic end, lying beneath the six lower left ribs, is in relation with the spleen and the gastro-splenic omentum, which contains the splenic vessels.

The œsophageal opening resembles an inverted funnel. It is fairly in the middle of the diaphragm, but on a plane posterior to the pylorus.

The **Duodenum** is from eight to ten inches long. It extends from the pylorus, round the head of the pancreas, to the left side of the second lumbar vertebra. It has no mesentery, and is the widest portion of the small intestine.

The first portion ascends to the liver and the neck of the gall bladder, having the hepatic artery, portal vein, and common bile duct behind it.

The second portion lies on the right kidney ; it is overlapped by the ascending colon. On its inner aspect it is separated from the head of the pancreas only by the common bile duct and the pancreatico-duodenal vessels.

The third portion has in front of it the stomach, the descending layer of the transverse meso-colon, and the superior mesenteric vessels. Behind are the crura of the diaphragm, the inferior vena cava, and the aorta, with the origin of the superior mesenteric artery. Its upper border is intimately in relation with the pancreas, the superior mesenteric vessels only intervening.

On its mucous surface we find valvulae conniventes and villi below the opening of the bile duct, and also solitary and Brunner's glands. The gut is invested with peritoneum only in its first and part of its second portions.

The **Small Intestine**, beyond the Duodenum, is about twenty feet in length. It is divided into Jejunum (upper two-fifths) and Ileum (lower three-fifths); but there is no sharp anatomical division.

It is suspended to the backbone by a long mesentery ; hence, the gut is extremely mobile, and is the more liable, therefore, to appear at the orifice of any abdominal incision. In the jejunum, the villi and valvulae conniventes are more frequent, whilst Peyer's patches are less numerous than in the ileum. The situation of Peyer's patches is opposite the attachment of the mesentery ; they run longitudinally to the gut, and are extremely vascular.

The ileum terminates at the ileo-cæcal valve, perhaps the most competent valve in the body. Vomiting of matters from the cæcum or large intestine does not occur, except the valve be injured or destroyed.

The **Large Intestine**, about six feet long, is subdivided into Cæcum, Ascending, Transverse and Descending Colon, Sigmoid Flexure, and Rectum.

The *Cæcum* is situated in the right iliac fossa. It is entirely surrounded by peritoneum. Behind, it rests on the iliacus ; in front, it touches the abdominal wall. The appendix, varying in length from three to eight inches, springs from the back part of the cæcum, and is directed upwards and inwards. It is surrounded by a fold of peritoneum.

The *Ascending Colon* passes in front of the right kidney to the under surface of the liver, then it takes a sharp, downward curve to gain the level of the transverse colon. This looping is important, as it favours the retention of fæces and flatus. This portion of the gut is bound to the back of the abdomen by a short fold of peritoneum or meso-colon.

The *Transverse Colon* courses in the folds of the great omentum, below the greater curvature of the stomach. As a general rule, its situation would be indicated by a horizontal line about three fingers' breadth above the umbilicus. Its course is from the under surface of the liver downwards, to gain the level of the lower border of the stomach, across the belly, and then it ascends to the spleen. Its upper border is, therefore, in relation with the liver and gall bladder and the lower end of the spleen. It lies some two inches from the great curvature of the stomach.

The *Descending Colon* extends from the spleen to the left

iliac fossa. It passes over the left kidney, the left crus of the diaphragm, and the left quadratus lumborum. In front, it has the convolutions of the small intestine. It is deeper and is less completely surrounded by peritoneum than the ascending colon.

The *Sigmoid Flexure* extends from the iliac crest to the sacro-iliac articulation. It has a complete meso-colon, and is extremely loose and movable.

The *Rectum* is eight inches long, beginning at the left sacro-iliac synchondrosis and ending at the anus.

The first portion lies on the sacrum, the pyriformis, the sacral plexus, branches of the left internal iliac artery, and the left ureter. It is entirely surrounded by peritoneum.

The second portion rests on the lower part of the sacrum and on the coccyx. In front is the trigone of the bladder, the prostate, and the vesiculæ seminales. Peritoneum invests the front and sides of its upper half.

The third portion touches the apex of the prostate, the membranous part and the bulb of the urethra, and then recedes from the urinary tract to emerge on the surface at the anus. The anus is guarded by a voluntary external sphincter; the internal (involuntary) sphincter is situated one inch higher up the gut, and above this again is a large dilatation or pouch on which the prostate rests.

In the female the relations are similar; but the uterus and vagina are immediately anterior to its second and to the upper part of its third portion.

THE ABDOMEN.—For clinical purposes the abdomen is divided into nine regions by two vertical lines which bisect two horizontal lines. There is no exact agreement amongst anatomical authorities as to where these lines shall be drawn, and in the following description we have observed, with slight modifications, the lines as given in Morris's 'Anatomy.' Thus, if one horizontal line be drawn across the belly at the lowest point of the costal border—that is to say, at the eleventh costal cartilage—and another at a level of the anterior superior iliac spines, we have three zones of latitude. Then if we draw two other vertical lines corresponding to the outer edge

of each rectus muscle, the abdominal surface is thus subdivided into nine regions : the middle ones, from above downwards, being known as *Epigastric*, *Umbilical*, and *Hypogastric* regions ; and the three lateral being named *Hypochondriac*, *Lumbar*, and *Inguinal* regions.

In the *Epigastrium* are found the liver, the pancreas (head and body), the spleen (upper part), the kidneys (upper and inner portions), the adrenals, the stomach, the duodenum, and coils of small intestine.

The *Umbilical* region contains the inner part of both kidneys and the ureters, the jejunum and ilium, the transverse colon, part of the sigmoid flexure, and the beginning of the rectum.

The *Hypogastrium* contains the small intestine, the sigmoid flexure, the rectum, the bladder in children, and in adults if distended, the uterus and its appendages.

The *Right Hypochondrium* contains the liver (greater part), the right kidney (upper third), and the hepatic flexure of the colon.

The *Right Lumbar* region contains the right kidney (lower two-thirds), the ascending colon, the cæcum, and some coils of small intestine.

The *Right Inguinal* region contains the inguinal canal, the cæcum (lower part), and coils of small intestine.

In the *Left Hypochondrium* are found the liver (left lobe), the spleen (outer part), the pancreas (tail), the stomach (great end), and the colon (splenic flexure).

The *Left Lumbar* region holds the left kidney (lower two-thirds), small intestine (especially jejunum), descending colon, and part of the sigmoid flexure.

The *Left Inguinal* region contains the inguinal canal, the small intestine, and part of the sigmoid flexure of the colon.

The umbilicus itself is on a level with the highest point of the iliac crest, and situated over the bifurcation of the aorta.

DISEASES OF THE MOUTH

STOMATITIS

Definition.—Inflammation of the mouth. Three different varieties are recognised at least, viz. : Parasitic, Ulcerative, and Gangrenous.

PARASITIC STOMATITIS (THRUSH)

This condition is most frequent at the extremes of life. It occurs in infants, especially those who are improperly fed and whose digestive powers are thereby weakened.

In old age it accompanies diarrhœa and the exhaustion of advanced phthisis, cancer, and other chronic, wasting diseases. Its appearance is often significant of approaching death. The exciting cause would appear to be the deposit and growth of a fungus (the *Saccharomyces albicans*), which is probably the same as causes acetous fermentation in wines.

Pathology.—The fungus consists of an interlacing mycelium with spores. It can easily be recognised microscopically by detaching a portion from a mucous surface and treating it with caustic potash on a glass slide. The organism first appears in the middle layers of the epithelium, and thence extends to the superficial and deeper layers. Each patch consists of epithelium, fat, and the fungus above described. In appearance they are white-looking, irregular-shaped spots like mould, slightly raised above the surface, with a zone of inflammatory tissue surrounding. On separation of one of these patches an abraded surface is left. They appear on the tongue, and mucous surfaces of the cheeks and buccal cavity, the secretions of which are acid, else the fungus could not flourish.

Although thrush is said to occasionally 'go through' the patient, it is probable that the anal erythema frequent in these cases is due to the diarrhœa which accompanies the disorder, the stools often being equally as acid as the buccal secretions.

Symptoms.—There are no special symptoms beyond some discomfort in the mouth, which is increased by taking food. The lips and tongue are dry ; often a ridge of heavy white fur is seen down the middle of the tongue, and the fungoid patches are found scattered about the mucous surfaces as described. Other signs which attend the disease are merely those of exhaustion and debility, which precede or attend the disorder.

Treatment.—Alkalies constitute a speedy and efficacious remedy. Carbonate of soda and aromatic powder of chalk in mixture act not only as astringents, but as antacids.¹ Local washes of chlorate of potash or borax in strong solutions may be applied to the mouth. An occasional laxative of castor oil is also advisable.

ULCERATIVE STOMATITIS

This form of stomatitis is characterised by the formation, almost invariably in young children, of ulcers, varying in size from a pin point to that of a split pea, on the mucous surfaces of the tongue, palate, and cheeks. The ulcers are sharply defined with irregular, elevated edges which form cup-shaped sores, the floors of which are covered by a thick, slimy, and often purulent secretion. At the same time the tongue is heavily furred, the breath foul, the bowels loose, and there is some extensive secretion and dribbling of saliva. Occasionally the disease extends to the sockets of the teeth, when the gums are swollen and tender and often the seat of hæmorrhage. These symptoms are accompanied by feverishness and irritability, by disinclination for food as well as difficulty in masticating it. The disease rarely occurs in adults ;

¹ R. Sodæ Bicarb: gr. iv.; Pulv: Cretæ Aromat: gr. v.; Spt: Ammonizæ Aromat: m̄v.; Aquæ Pimentæ ad ʒij. M. ft. mist.

and in children it is seldom a primary condition, being generally symptomatic of failing health and malnutrition.

Treatment.—As in parasitic thrush, alkalies are the best remedies. The bicarbonate of soda or potash may be given in five-grain doses every four hours. Solutions of chlorate of potash and of borax are also useful as gargles or washes. Decoction of cinchona may also be given. It not only improves the appetite, but acts as a general tonic. At the same time food should be nourishing and easily digestible. Milk had better be boiled, and diluted with lime water.

GANGRENOUS STOMATITIS (NOMA)

This condition occurs especially in young children mostly under five years of age. It is frequently a sequel of some specific fever, notably measles ; but may occur without any premonitory disease. It is probable, however, that a low state of health, deficient food, and defective sanitary surroundings are the most potent predisposing causes.

Symptoms.—During convalescence from, say, measles, our attention may be drawn to the mouth by fœtor of breath. On examination of these parts an unhealthy-looking ulcer will be found inside the cheek or lips, with a greyish, foul slough forming its floor. There is not necessarily any attendant pain ; the condition may even have existed some days before attention is drawn to it. The ulceration is usually accompanied by salivation and perhaps hæmorrhage, by marked gangrenous odour, and by enlargement of the neighbouring lymphatic glands. Grave constitutional symptoms may also be present. The pulse is rapid and small ; there is slight feverishness ; and, although the appetite continues good, marked signs of asthenia and collapse are apt to supervene rapidly, during which the patient expires. Should recovery take place, it is at the cost of permanent and extensive disfigurement, owing to loss of tissue. In some cases the disease stops at perforation of the cheek or lip ; in others it extends rapidly until the whole of one side of the buccal cavity, or even the adjacent bones become eroded.

Treatment.—Plenty of good nourishing food, and stimulants are required. The disease must always be regarded as one tending to destroy life, not so much from the effects of the local destruction as from the grave constitutional exhaustion it causes. Opiates are usually required to afford rest and to relieve pain. The local treatment of the gangrene is mainly surgical. The wound should be dressed antiseptically, and kept aseptic as much as possible. Corrosive sublimate dressings, however, are better avoided. Strong solutions of carbolic acid are better. They relieve fœtor, and have a slightly anæsthetic action. The extension of the gangrene is often arrested by the application of strong nitric acid, hydrochloric acid, nitrate of silver, or even the galvano-cautery to the sore and parts around.

GLOSSITIS

Definition.—Inflammation of the tongue.

Causation.—It is usually caused by some direct injury, such as a fall on the face when the tongue happens to be protruded ; or it may be produced by the bites or stings of irritant insects. It not infrequently is excited by the use of mercurials and other drugs. It occurs occasionally in syphilis, apart from gummatous induration.

In some few instances it is idiopathic.

Symptoms.—The tongue becomes stiff, swollen, and painful ; all movements of the organ are difficult and aggravate the pain. Articulation is performed only with an effort, and is then indistinct. As the disease advances, the tongue becomes greatly enlarged ; it protrudes itself from the mouth, and is indented and often excoriated by the teeth. Mastication is next to impossible ; saliva collects in the buccal cavities, and overflows by the angles of the mouth. The tongue, after a time, becomes heavily coated with a thick fur and slimy mucus, and there is usually some fœtor.

In most instances the inflammation subsides within a week, and the organ assumes its normal size and condition. In rare cases, however, it remains permanently hypertrophied, and requires surgical treatment ; but a more frequent change

than this is suppuration. This is attended by some feverishness and rigors, together with a sensation and fear of impending suffocation. The latter danger, indeed, may be real, from inflammatory extension from the tongue to the epiglottis and larynx.

Treatment.—Give liquid nourishing food, such as beef-tea, eggs, milk, &c. Alcoholic stimulants are not required, and they would irritate the tongue whilst passing over it. Ice to suck may be freely allowed. Antiseptic and astringent washes should be constantly used. The tincture of the perchloride of iron appears to possess both these qualities, and in many instances acts beneficially in a surprisingly rapid way. Deep incisions may be made along the dorsum of the tongue if the enlargement be rapid. Tracheotomy may be required if the inflammation extend to the air-passages.

TONSILLITIS (QUINSY)

Definition.—Inflammation of the tonsils. One or both glands may be affected at the same time.

Causation.—(a) **PREDISPOSING.**—*Age.*—It is most frequent between twelve and eighteen, at a period when there is the greatest strain on nutritive processes. *Sex* has apparently no predisposing influence. *Climate.*—The disease is most rife in cold and damp countries. *Rheumatic Diathesis.*—It frequently attacks those who have previously suffered from, or who have a predisposition to, rheumatism. *A Debilitated Constitution*, or any illness by which the patient is brought below par, also acts as a predisposing cause. It thus supervenes on catching cold after severe muscular exercise.

(b) **EXCITING.**—Some apparently specific and septic material emanating from cesspools, or defective drainage, or from offensive discharges in surgical wards of a hospital. It may also be directly propagated from one subject to another by the exhalations from mouth and nose of a previously affected person. The contagious character of the disease is most pronounced at the beginning of the illness.

Pathology.—The earliest stage is characterised by catarrh of the mucous membrane of the tonsil, which is not limited to the surface, but extends to the follicles and to the soft palate and uvula. As a result, there is at first an arrest of secretion, followed by a somewhat copious discharge which forms on the surface a greenish, sticky film which can be easily removed by scraping. Occasionally in rheumatic subjects the inflammation extends to the planes of connective tissue in the tonsil, and to similar planes in the pharynx and soft palate. The inflammatory process lasts two to three days, when (1) resolution takes place, the exudation being washed away or expectorated ; or (2) suppuration takes place, owing to cellular proliferation being so extensive as to interfere with the nutrition of the gland. The interior then breaks down, and an irregular abscess cavity is formed (Quinsy). This condition appears to be rarer than one would suppose, and mostly occurs in those who have feeble constitutions. (3) A third termination of acute tonsillitis is of the nature of chronic, inflammatory change characterised by hypertrophy of the gland, especially of its interstitial connective tissue. The tonsil then becomes painlessly enlarged, it loses its natural pink colour, and assumes a paler tint ; the uvula is pushed on one side, and the faucial and respiratory passages are considerably obstructed.

Symptoms.—Vary somewhat as the disease runs an *Acute*, *Sub-acute*, or a *Mild* course.

An *Acute* case is generally ushered in with pain and stiffness in the neck, a dryness of the throat which necessitates frequent swallowing of saliva, and severe headache. This last symptom is usually constant and severe. In a few hours the patient further complains of rigors, uneasiness, and pains in the bones and muscles, and a total loss of appetite. On examination at the end of twenty-four hours there is a high fever (103°) ; the pulse is full and quick (110 to 120) ; the urine is scanty and febrile, with occasionally a tinge of albumin when the temperature exceeds 103° ; the tongue is loaded with a thick, heavy, and slimy fur ; the bowels are constipated ; and the breath is foul. On inspecting the fauces,

one or both tonsils will be found enlarged and exuding a greenish lymph from their follicles, or a layer of similar exudation is smeared over their surfaces. The glands at the angles of the jaws are swollen and tender ; deglutition is only carried on with pain, difficulty, and grimace ; the speech is characteristically thick ; and the expression of the face is also peculiar and almost diagnostic. It is one of pain, weariness, and sadness : the angles of the mouth being drawn outwards and downwards, and the forehead transversely wrinkled. This condition lasts three days, when a crisis occurs, either in the form of a profuse sweat, or a copious diuresis, or a diarrhœa ; it is accompanied by an abrupt fall in the temperature to almost the normal line, and the patient feels comparatively well.

If the crisis does not occur on the third day, the case will probably, according to Dr. Haig-Brown, go on to suppuration. He insists, however, that this only occurs in feeble and unhealthy subjects. In suppuration the dysphagia and the febrile conditions continue ; the tonsil becomes red and tense ; there is fear, generally quite groundless, of suffocation, till about the sixth day, when the abscess points, usually towards the mid-line, and discharges itself by a ragged, sloughing aperture.

In *Sub-acute* cases the fever and the symptoms generally are less severe : the temperature only rises to 101° or 102° ; the throat is less painful ; there is less follicular discharge ; the general bodily weakness is not so marked ; and the patient is usually convalescent on the third day.

A *Mild* attack is characterised merely by an irritable sore throat, the tonsils being only red and swollen, by slight headache, and a temperature of 99° to 100° .

Dr. Haig-Brown, however, draws attention to the clinical fact that these milder cases are quite as infectious as the severer forms.

In all varieties the disease may recur ; or it may be metastatic, in that one tonsil may take on the inflammatory changes just as its fellow is healing ; and it does not appear unlikely that in many cases it is a manifestation of one of the

many different forms of rheumatism which are peculiar to young people.

Complications.—As a rule, none. 1. **AURAL.**—Deafness may ensue, owing to mechanical obstruction of the Eustachian tube or to inflammatory extension to its mucous membrane. In unhealthy subjects the inflammation may extend to the middle ear, causing perforation of the membrana tympani, or even fatal meningitis.

2. **CARDIAC.**—Any form of endocardial or pericardial inflammation may supervene. Lesions of the mitral valve, especially mitral regurgitation, are the most frequent.

3. **ACUTE RHEUMATISM.**—This disease, when it does occur as a complication, commences about the fourteenth day. It is by no means common ; but attention may be here drawn to the apparent clinical relations which exist between tonsillar and joint inflammations. This is seen in scarlet fever, in septic sore throat, as well as in tonsillitis. Further, many cases of acute rheumatism commence with symptoms of tonsillar and faucial inflammation.

4. **RELAPSE.**—The tonsil may become again inflamed, and run through a course similar to the initial attack. Occasionally there are second or even third relapses.

5. **CHRONIC HYPERTROPHY OF THE TONSILS.**—This occurs in young and old, especially those who inherit a strumous taint. The gland is enlarged and hardened, so that deglutition is permanently difficult ; respiration is impeded ; the voice is nasal ; speech is defective, especially when the hypertrophy commences in early life ; and there is a permanent characteristic expression of face similar to that described as occurring during the acute stage. We are also convinced that the chronic hypertrophy exercises a baneful effect on the mental development of the patient.

Diagnosis.—(a) In **SIMPLE CATARRH OF THE THROAT** the tonsils are merely red ; there is no follicular exudation ; no tenderness at the angles of the jaw ; and the pyrexial symptoms are less pronounced.

(b) **FROM SCARLET FEVER.**—Here we are guided by the early presence of rash ; a ‘strawberry’ tongue ; and a temperature

of 103° to 104° , or higher. In addition, we find a scarlet blush invading palate, fauces, and pharynx.

(c) In RÖTHELN there is no follicular tonsillitis ; the throat is merely dry and red ; the characteristic eruption appears on the second day, followed by 'branny' desquamation in the second week.

(d) FROM MEASLES.—In this fever we are guided by coryza, sneezing, catarrh of the respiratory passages, and rash. The critical fall of temperature does not occur till about the eighth day.

(e) FROM DIPHTHERIA.—In this disease we find a false membrane in ashy-grey patches, of a tough leathery character, and leaving excoriations on removal. The pallor and exhaustion are pronounced, albuminuria is an early symptom, and is commensurate with the extent of the exhaustion.

Treatment.—(a) GENERAL.—Isolation is necessary ; all the crockery and spoons used by the patient should be properly cleansed and disinfected. A medicated spray may with advantage be used, not only in the room, but to the patient's throat. Perfect rest in bed should be ordered for four days, at least. The diet should be good and nutritious, but entirely fluid, owing to the extreme difficulty in deglutition. A preliminary saline purge should be given.

(b) LOCAL.—No medicines appear to have any curative effect, nor do any appear to have any influence in arresting the disease. Guaiacum, aconite, chlorate of potash, all have their advocates. We have certainly seen the best results from guaiacum (mist. guaiaci or trochisci guaiaci B.P.) and salicylate of soda (gr. x.), especially in rheumatic subjects. Locally we may use astringent washes or lotions, and here a strong solution of salicylate of soda applied with a mop brush is of great use. Ice to suck is very soothing. Gargles are better avoided ; they are difficult to manage, and often cause pain. Inhalations of steam appear to give the most relief. Poultices or hot wool-packs may also be applied with advantage to the parts behind the vertical ramus of the jaw ; they should be tied up to the head like a bonnet-string, and not round the neck like a collar. An excellent method

of applying heat is a split baked potato ; it retains its moisture and heat for a considerable time. After the fever has abated the patient will require tonics (quinine and iron) and a generous dietary.

Examine the patient carefully from day to day to ascertain the possible existence of ear or heart complications. If suppuration supervene, do not be in a hurry to apply the bistoury. The pus soon finds a ready outlet, and there is rarely any danger from suffocation.¹

¹ See Dr. Haig Brown : *Tonsillitis in Adolescents*.

DISEASES OF THE ŒSOPHAGUS

The principal diseases of this canal which come within the cognisance of the physician are Inflammation, Stricture and Obstruction, Uleeration, and Dilatation.

Inflammation of the Œsophagus

This condition is very rare as an idiopathic disorder. It is usually secondary to the injuries caused by drinking hot fluids, corrosive acids, such as the hydrochloric and other mineral acids, also carbolic and oxalic acids. The swallowing of corrosive acids is nearly always a suicidal act. Inflammation due to scalding not infrequently occurs in children, who have been known to drink from a kettle's spout.

Beyond these causes, œsophagitis may be caused by the pressure of abscess, tumours, or other new growths springing from glands in the neighbourhood of the canal.

In those cases which recover, the inflammation is apt to produce warty excrescences within the tube, which again may give rise to symptoms of obstruction.

Occasionally a secondary varicosity of œsophageal veins occurs, which may give rise to serious hæmatemesis.

Stricture and Obstruction of the Œsophagus

The causes which may give rise to this condition are numerous and varied. The following table is an enumeration of those most frequently met with

Stricture and obstruction	{	(a) Fibrinous stricture.
		(b) New growths { Malignant. Syphilitic.
		(c) Spasm.
		(d) Paralysis.
		(e) Tumours of surrounding structures.
		(f) Impacted food, false teeth, or other foreign body.
		(g) Simple ulceration.
		(h) Dilatation.

Fibrinous Stricture may be due to a chronic local thickening or cicatricial tissue, subsequent on some injury to the tube.

Malignant new growths are most frequently epitheliomatous in their character; but scirrhus and other forms of cancer are not unknown. Their most frequent situation is opposite the bifurcation of the trachea, and they are almost always primary manifestations of malignant disease. Ulceration occurs sooner or later in all malignant strictures.

Syphilitic lesions are tertiary manifestations. Occasionally a gummatous tumour forms in the walls of the canal, and forms an obstruction; but more frequently the stricture consists of cicatricial tissue, as in similar lesions in the rectum.

Spasmodic stricture occurs in hysterical women and other neurotic subjects. In these individuals the symptoms and complaints of suffering outweigh the extent of the physical signs, the obstruction to food being always overcome by passing an œsophageal tube. In addition, the symptoms may suddenly 'become cured,' to reappear at some future date.

Stricture—or rather obstruction—due to *Paralysis*, again, is found in hysterical women, unless, as may be the case, it is due to some gross lesion in the medulla oblongata, or secondary to the effects of inflammation of the tube itself.

Obstruction due to *Tumours* originating in organs or parts in the immediate neighbourhood of the œsophagus, forms a very wide subject. Keeping in view the anatomical division

of the œsophagus into cervical and thoracic portions, we may also group the causes of external obstruction of the œsophagus into those originating in the neck, and those in the thorax.

Cervical obstruction may be caused by enlargement of the thyroid body, abscesses in the cervical planes of fascia, aneurysm of the common carotid arteries, exostosis of cervical vertebræ: these are the most common.

In the thorax, œsophageal obstruction immediately suggests aneurysm of the aortic arch, or of the descending aorta, or of one or other of the large trunk vessels given off from the arch. It may, however, be caused by new growths originating in the mediastinal glands or areolar tissue, by abscess, or by great distension of the pericardium by fluid. One or two cases have been recorded in which obstruction was caused by hydatid cysts. Whatever be the cause in this category, we are guided, not only by dysphagia, but by the signs which are described under Aortic Aneurysm, Malignant Growth of the Mediastinum, Pericarditis, &c.

Obstruction due to the *Impaction* of imperfectly masticated food, or a plate of false teeth, needs no detailed description—the accident is so sudden and alarming, and the history so evident.

Simple Ulceration of the œsophagus is usually a secondary condition either to direct injury or to some of the preceding causes. The ulceration may be extensive, and completely surround the gullet, or it may be a local abrasion extending more or less deeply into the coats of the tube.

Dilatation, again, is usually a condition consequent on obstruction, although it is itself a cause of obstruction. It occurs above the seat of the original constriction, and may reach to large, pouch-like dimensions in which food is partially digested by saliva, and from which it can be regurgitated almost at will.

Symptoms.—In all cases of œsophageal obstruction *Dysphagia* is the most marked symptom, and one common to all. So important is it that in some treatises it has been especially described almost as a disease itself. The difficulty in swallowing is referred to one spot, the patient placing his finger on one

or other place in the neck or on the front of his chest. Singularly, the sense of obstruction does not appear to be referred to the vertebral region, as one would expect it should be from the nearness of the gullet to the backbone. The seat of the obstruction as felt by the patient, however, is no reliable guide to its actual site.

Dysphagia is first noticed in swallowing solids. A morsel of cheese or a piece of pie-crust appear to be especially difficult to pass. After a time, minced food and soft puddings are managed only with difficulty, until eventually—but in rare instances only—the obstruction is impervious to fluids even.

In some conditions the dysphagia is very sudden in its onset, *e.g.* in acute inflammation of the œsophagus ; in others, such as aneurysm or cancer, the difficulty supervenes gradually. Moreover, in aortic aneurysm dysphagia frequently abates and recurs, according to the extension of the sac in different directions ; so that dysphagia is by no means a constant sign. In hysterical obstruction, dysphagia also comes and goes ; but a bougie passes without difficulty, and cures the symptom. There is, in fact, no true obstruction. The obstruction in fibrinous stricture progresses to a certain degree only, and then stops.

If the obstruction be complete, regurgitation of food which has accumulated above the constriction occurs sooner or later ; the period after food at which this takes place depending on the site of the obstruction. The higher up the gullet that the stricture exists the earlier the regurgitation ; a similar remark applying to obstruction of the bowel.

Results of Obstruction.—In all cases emaciation occurs, but the rapidity of the loss of flesh varies according to the cause and character of the obstruction, and the amount of food which it allows to pass. In the cancerous stricture, for example, not only is food sparingly admitted to the stomach, but the cachexia of the disease is also in evidence.

Occasionally perforation occurs, especially in malignant or other ulcerations, thus forming a fistulous communication with the posterior mediastinum, the air-passages, or with some large vessel.

In nearly all cases the mental faculties remain clear to the very last, the patient being quite cognisant of his terrible position. But beyond this there is a mental anxiety which is intense and almost characteristic in all cases of œsophageal obstruction, whether it be temporary or permanent.

Treatment.—Ascertain the situation, extent, and tightness of the stricture by carefully passing an œsophageal bougie, measuring its exact position from the incisor teeth. It is necessary to point out that this operation should only be performed by skilled hands ; that it is one of danger in advanced malignant ulceration ; and that if there be evidence that the obstruction is due to aortic aneurysm, it is altogether unnecessary. Occasionally the approximate situation of the obstruction may be revealed by carefully auscultating the back in the course of the œsophagus. In partial obstruction, fluid may be heard to trickle through the stricture, instead of passing down in a gulp, as in health.

In simple stricture and in hysterical paralysis of the gullet the passage of graduated bougies is generally sufficient to effect a cure.

In impervious strictures nutrient enemata may afford a temporary respite. Gastrostomy may be required in some instances in which the stricture, although almost complete, is not malignant, but one which is likely to yield to prolonged dilatation.

If gastrostomy be decided upon in malignant stricture, the sooner it is performed the better. There comes in malignant disease a day, which no one can foretell, on which the patient suddenly changes for the worse, and is beyond all help.

DISEASES OF THE STOMACH

HÆMATEMESIS

Definition.—Vomiting of blood.

Causation.—Hæmorrhage may proceed : (a) FROM THE ŒSOPHAGUS, owing to malignant ulceration, varix of œsophageal veins, injuries.

(b) FROM THE STOMACH.—The hæmorrhage may be caused by some breach of membrane, as in ulcer (most common), new growth, the action of corrosive acids, rupture of an aneurysm ; or it may be due to passive obstruction to the portal circulation, as in cirrhosis of liver ; to disease of the capillaries (lardaceous diseases) ; to the effects of injuries, blows, strains, or the result of acute inflammation, of over-eating and -drinking. It often attends certain specific fevers (yellow, typhus, and malarial). Finally, it occurs in various altered conditions of the blood itself (scurvy, purpura, syphilis). It has also been attributed to the suppression of habitual discharges, and to vicarious menstruation, but probably without good clinical evidence.

(c) FROM THE DUODENUM.—In portal congestion, or as a result of corrosive acids, and of ulceration secondary to burns and scalds.

Whatever be the site and the cause of the hæmorrhage, it is more frequent in women than in men, and during middle life than at any other period.

Symptoms. Vary, according to the amount of the hæmorrhage. If the bleeding be free, the first sign may be sudden pallor and syncope. Ordinarily, however, hæmatemesis is preceded by a sense of weight at the epigastrium, and feelings

of nausea and giddiness. The vomited matter may at first consist of mucus and partially digested food, followed by hæmorrhage as a direct result of the effort of vomiting ; or, on the other hand, the exuded blood acting as an irritating substance may directly cause the vomiting. Thus the hæmorrhage will vary in amount, according to the extent of the injury or the size of the blood-vessels involved ; it may vary in colour and consistency, according to the length of time the blood has remained in the stomach, and be thus altered by digestive processes.

Inspection of the stools is important ; they are not unfrequently black (*melæna*) from passage of the blood into the duodenum, or from ulceration of the duodenum itself. In addition, the patient also presents a degree of anæmia variable according to the amount of blood lost ; the tongue is coated, digestion impaired, and the breath often obtains a characteristic foul odour.

Prognosis.—As a rule, favourable. The danger is not so much in the hæmorrhage as in its cause. Death seldom occurs as an immediate result of hæmatemesis, unless it be due to rupture of some large vessel.

Diagnosis.—Inspect the mouth, nose, fauces, and pharynx (as in hæmoptysis). Blood may issue from any of these channels and be swallowed. From hæmoptysis it is diagnosed by (1) the blood being vomited, and mixed with food and the secretions from the stomach, and therefore of acid reaction ; (2) the absence of cough and the physical signs of pulmonary disease ; (3) the history of gastric disturbance ; (4) the presence of *melæna* ; (5) the age of the patient (middle life).

Treatment.—Absolute rest. The food should be light and easily digested, as in the treatment of gastric ulcer. Milk, farinaceous foods, beef-tea are best. Ice, or iced water may be given, but avoid alcohol and warm fluids. Further treatment must depend on the cause of the hæmorrhage. Drugs are not of much service. Still opium may be given, on account of its topical and general effects ; or it may be combined with acetate of lead. Beyond these, vegetable astringents may be given (*kino*, *catechu*, and *tannic acid*). The best results, how-

ever, seem to follow the hypodermic injection of ergotin. It is often advisable in portal obstruction not to arrest the hæmorrhage, but to relieve the congested venous system by light purgatives.

GASTRITIS

Definition.—An inflammation of the stomach, usually commencing in the mucous membrane. It may thence extend to the other coats.

Three degrees of gastritis are recognised: ACUTE; SUB-ACUTE; CHRONIC.

Causation.—The ACUTE form is rare except as the result of some irritant poison, such as a corrosive acid, or putrid food. It may, however, be caused by copious libations of very cold water. The SUB-ACUTE and the CHRONIC varieties may be brought on by over-feeding, by hard, indigestible foods, and strong alcohol; or they may be secondary to acute fevers, to cirrhosis of the liver, and Bright's disease.

Pathology.—ACUTE FORM.—The stomach is contracted; its mucous coat is wrinkled and softened, often presenting ecchymoses and extravasations of blood, with, it may be, in addition, large irregular ulcerations, on the floors of which sloughing patches are seen.

SUB-ACUTE.—The post-mortem appearances are not so marked as in the acute form. The capillaries of the mucous membrane are injected, especially near the pylorus. There is also a secretion of much ropy, alkaline mucus. The stomach is not unfrequently slightly distended.

CHRONIC.—The mucous membrane is thickened, pale, and wrinkled; in it are patches of pigmentation from old hæmorrhages. By the microscope it is found that there is atrophy of the peptic glands, fatty degenerative change in their epithelium, with increase of the intertubular connective tissue. The organ is usually more or less distended, owing to atrophy of its muscular coat.

Symptoms.—ACUTE GASTRITIS.—The most marked symptom is pain in the epigastrium of a burning character

accompanied by marked tenderness. In addition, we find rigidity of belly wall; vomiting which increases the pain (the vomit consists of mucus and perhaps blood), hiccough, fever, and thirst; the tongue is foul; the bowels are constipated; there is marked mental distress and anxiety; the pulse is small and quick; respiration is hurried and chiefly thoracic; the skin is cold and clammy. After a time these symptoms gradually subside; the disease may, however, become chronic, or terminate in death from collapse.

SUB-ACUTE GASTRITIS.—Many symptoms of same character as in the acute stage. Thus there are pain, nausea, vomiting, which are all increased by food; tenderness over the stomach, with general epigastric and interscapular distress; the bowels are irregular; the urine concentrated, scanty, and contains lithates. The temperature does not vary much from normal.

CHRONIC GASTRITIS.—Dyspepsia is the most prominent feature; it may indeed sum up the whole disease. The tongue is glazed or deeply fissured, the gums are spongy, the breath is foul, and aphthæ are common. In addition, it is found that there is thirst, complete loss of appetite for healthy food (although craving for fruit), and vomiting, at longish intervals, of large quantities of bile-stained mucus. The vomit may contain blood, but hæmatemesis is not prominent. In addition, the bowels are costive; the patient may also have piles, a darkened areola round the eyes, and other signs of sluggishness of the portal circulation. The mental state is likely to degenerate into extreme irritability or hypochondriasis.

Treatment.—**ACUTE GASTRITIS.**—The first indication is to apply the antidote to the poison, if the disease be due to one. No food is necessary, as it cannot be taken. We may, however, allow ice to be sucked, or cold milk and water to be sipped. Opium in full doses is the only remedy. It may be given by mouth, or better still as morphia by hypodermic injections. Maintain the strength by nutritive enemata.

SUB-ACUTE GASTRITIS.—Apply sinapisms or hot packs to the epigastrium. The food should consist of milk and weak

animal broths. Alcohol should be prohibited. The bowels must be regulated by non-irritant laxatives (carbonate of magnesia, castor oil). As regards drugs, favourable results are obtained by bismuth, alkalies (especially in the effervescent form), hydrocyanic acid.^{1, 2, 3.}

CHRONIC GASTRITIS.—Attend carefully to the diet; the patient's own experience as to what agrees with him is a good guide. Forbid alcohol. Order gentle exercise. There should be a daily action of the bowels; and even then an occasional large enema (soap and water, and olive oil) will bring away much accumulated fæces. For the recurrent dyspepsia give bismuth, alkalies, gentian, and quassia. Creasote (m.j.) three times a day, or a similar dose of liq. arsenicalis frequently relieves the distressing vomiting. Calumba as a stomachic is better when there is much pain. Rouse the portal circulation with saline purges and an occasional small dose of calomel.

GASTRIC ULCER

Definition.—A primary benign ulceration of the mucous membrane of the stomach. Two varieties are recognised: 1. the superficial, spreading, 'smouldering' ulcer; 2. the perforating ulcer.

Causation.—Still obscure in both varieties. In the superficial form the ulceration appears to be connected with alcohol, over-feeding, irregular method of taking food, or other circumstances which tend to derange the portal circulation. It is more common in males, especially after the prime of life.

The perforating ulcer occurs in very large proportion, in females between puberty and thirty. According to Virchow, it may be due to obstruction of vessels of a given area of the stomach by embolism or thrombosis, leading to death of the

¹ R̄. Bismuthi Carb: gr. v.; Magnes: Carb: gr. x.; Acid: Hydrocyanici Dil: mīij.; Mucil: Tragacanth: ʒj.; Aquam ad ʒj. Misc.

² R̄. Sodæ Bicarb: gr. xv.; Acid: Hydrocyanic: Dil: mīij.; Spt: Annon: Aromat: m̄xv.; Infusi Gentianæ Co: ad ʒj.

³ Potass: Bicarb: gr. xx.; Acid: Hydrocyanic: Dil: mīij.; Tinct: Lavand: Co: m̄xv.; Aquam ad ʒj. Ft: Mist: Alk.

R̄. Acid: Citrici gr. xv.; Aquam ad ʒj. Ft: Mist: Acid.

part ; but in view of the splendid collateral circulation of the stomach, this is difficult to accept. According to Wilks, the ulceration is a neurosis analogous to simple ulcer of the cornea. It is almost invariably preceded by constipation and chlorotic anæmia ; and it appears to us that gastric ulcer may be the climax of these prior conditions, and not so much a primary affection itself, since similar conditions are not infrequently observed in the rectum after chronic constipation. Dr. Ord has drawn attention to its association with organic heart disease (endocarditis and pericarditis). (Medical Society, Dec. 1891.)

Pathology.—1. The superficial ulcer varies in size from a small nut to that of a man's palm. The edges are irregular in outline and thickened, owing to an inflammatory zone surrounding them. As a rule, the edges slope down to the floor of the ulcer, which may be formed by any one of the tunics of the stomach, according to the depth of the erosion. The floor itself may be smooth, it may be irregular from the presence of sloughing tissue, or it may be actually formed by some underlying organ or vessel. It is not unusual to find one or more patches of erosion, with intervening tracts of healthy mucous surface.

2. The perforating ulcer is small, generally the size of a sixpenny-piece, clean cut, with perpendicular and hard edges. It may also involve any of the coats of the stomach and perforate into the peritoneal cavity, or be arrested by pancreas or liver to which the stomach has been previously glued by inflammatory exudation. The situation of the ulcer is usually on the posterior wall (40 to 50 per cent.), near the upper curvature, and perhaps nearer the pyloric than the cardiac end. It is surrounded also by a girdle of induration. In the process of healing, the floor is filled by opaque, cicatricial tissue, the surrounding induration diminishes, but leaves a concentric puckering from which may result contraction and gross deformity of the stomach.

Symptoms.—1. In the *Superficial Ulcer* the symptoms are mainly those of chronic catarrh with sub-acute inflammation, viz. : epigastric pain, increased by food ; a tongue which is irritable, and red at the tip and edges ; vomiting, the vomited

matter often consisting of huge quantities of mucus intermixed with blood ('coffee-ground vomit'), especially if there be attendant portal congestion. There is anorexia, or it may be, at times, a huge and depraved appetite; together with emaciation, progressive prostration, and death, unless the condition be relieved.

2. The *Perforating Ulcer* has somewhat similar symptoms. Before symptoms become pronounced there is constipation, a characteristic pallor, flatulence and uneasiness after food, epigastric tenderness, and pyrosis. When established, it is marked by three cardinal symptoms, viz. : (i) pain, which is increased by food; (ii) vomiting which relieves the pain; and (iii) occasional vomiting of blood.

In addition to the above we find the stools are often of a tarry character (melæna), owing to blood passing into the intestines; the pain, nearly always present, frequently extends to the intrascapular region: it is of a distressing, gnawing character, and has marked exacerbations, especially when food reaches the ulceration. The expression of face is anxious, dejected, and careworn. The disease may last for years; it may be temporarily arrested or cured, and recur again at some near future date. It is almost invariably preceded by amenorrhœa or leucorrhœa.

It is a question with us whether the perforating ulcer is a primary stomach disease, or whether it is not a secondary manifestation of some neurosis, and is, therefore, the crisis of some disorder as much as herpes is. (See Chlorosis, p. 479.)

Prognosis.—Generally favourable in both varieties. This will depend, however, upon method of living, and ability to obtain rest and careful nursing. Death may occur from (i) perforation and general peritonitis, (ii) profuse hæmorrhage, (iii) exhaustion. It should always be remembered that ulceration may exist, although the symptoms are in abeyance; and death may hence occur from sudden perforation or from hæmorrhage.

Diagnosis.—It is distinguished from Gastralgia by the local pain occurring after food, and its frequent occurrence in anæmic young women. It may simulate Cancer of the

stomach. The following table gives the chief points of diagnosis.

—	Perforating Ulcer	Cancer
Usual site.	Posterior surface; lesser curvature; near pylorus	At pylorus, or cardiac end
Tumour	None	Usually to be felt
Age	Between puberty and thirty	After middle life
Sex	Females	Males
Pain	Gnawing; intermittent; increased by food	Lancinating; constant; not immediately affected by food
Vomiting	Directly after food	Some time after food: it may be three hours
Hæmatemesis	Abundant; 'coffee-ground' character, but not so frequent	Scanty; occasionally scarlet-coloured; and frequent
Duration	Often chronic	Short; death in twelve months
Prognosis	Favourable; death, if it occurs, usually due to perforation or hæmorrhage	Unfavourable; death by exhaustion

Treatment.—1. *Arrest the Vomiting and Pain.*—For vomiting give alkalis (bicarbonate of soda or potash), especially in effervescent form; dilute hydrocyanic acid; oxalate of cerium; ice to suck. Apply sinapisms, poultices to the epigastrium, or blisters even, in severe cases. For pain, opium is the best remedy, in small continued doses to its full physiological effect: it has thus a topical influence, and is better than morphia subcutaneously. Bismuth (the subnitrate or the carbonate) may be added, or given alone. Nitrate of silver¹ has also been advocated, especially in chronic cases. It is 'inferior to bismuth, and should be given cautiously and not in a prolonged course. 2. *To control Hæmorrhage.*—Opium is the best remedy usually, but it may be supplemented by tincture of hamamelis (℥xx. in hot water), or by subcutaneous injections of ergotine, in severe cases. 3. *To*

¹ R. Argent: Nit: gr. $\frac{1}{6}$; Unguent: Kaolin: q.s. Ft: Pil.

promote Healing.—Diet is the principal factor. Give the stomach as much physiological rest as possible. If milk can be tolerated, administer it in half-wineglassful doses every half-hour ; lime water (one part to three) may be added if vomiting be urgent ; or the milk may be previously peptonised. In severe cases it is best to nourish the patient by enemata entirely.

As symptoms subside, farinaceous foods may be given (Benger's food, rice, sago), or some light, easily-digested animal food (sweetbread, tripe, raw meat juice). The value of all this dietary, however, will be greatly influenced by the taste and caprice of the patient. All food should be given cold or nearly so. Avoid tea, coffee, and alcohol in all forms. Nor is beef-tea advisable, as it is not well tolerated ; and when vomited it has some resemblance to hæmatemesis, and may mislead. For similar reasons the preparations of iron should be avoided until complete healing has taken place. 4. *To prevent a Recurrence.*—Attend to the general health. The diet should be light, wholesome, and easily digestible. Avoid hard and salt meats, pastry, cheese, uncooked vegetables, pickles. The meals should be small and at short intervals. Encourage gentle exercise in the open air, or at some bracing seaside resort. It is also of prime importance to regulate the bowels, either by large daily enemata, or by castor oil, pulv. glycyrrhizæ comp., pulv. rhei co., pulv. hyd. c. cretâ., or some simple laxative. Violent irritant purgatives are especially injurious. For the attendant anæmia small doses of iron are best, either in the form of the saccharated carbonate or the reduced iron. Arsenic is not to be recommended, owing to its irritating quality.

MALIGNANT DISEASE OF THE STOMACH

Definition.—A general term applied to cancer or other malignant new growth involving the stomach or pylorus. It is usually a primary affection.

Causation.—Nothing is known as to exciting causes beyond those commonly ascribed to all cancerous affections. It seems to affect those parts of the stomach (pylorus, cardiac

end) which are susceptible to irritation or injury from pressure of food. It is more common in adults than simple ulcer, and frequent between forty and sixty-five years of age.

Pathology.—All forms of malignant new growth are common in the stomach ; scirrhus is most frequently met with, then medullary and colloid in the order named : occasionally two varieties of cancer are met with in the same subject. The pylorus is the most frequent site, probably owing to irritation caused by the passage of undigested food ; but the new growth may also commence in the lesser curvature. Wherever it starts it has a tendency to form an annular constriction of the organ.

In **SCIRRHUS CANCER** the disease commences in the fibrous coat, then extends to the peritoneal tissue, which becomes thickened and adherent to neighbouring parts. It thence spreads to the muscular coats, causing them (i) to apparently increase in bulk, this being due to the infiltration of fibrous bands between the various muscular bundles ; and (ii) to undergo fatty degeneration. The mucous membrane is swollen, and projects above the surrounding surface. It eventually breaks down and ulcerates, leaving an uneven, fungating sore with thickened, everted edges, and a floor that is irregularly granular, or covered with slough.

In **MEDULLARY CANCER** the disease may commence either in the serous or in the fibrous coats. The growth is characterised by early induration followed by rapid extensions, until a large, lobulated, hemispherical tumour is formed, which ulcerates and sloughs with great rapidity, leaving a surface which is often studded with secondary deposits or with villous projections, rich in blood-vessels. These rapid excavations, therefore, are often attended by copious hæmorrhages.

When the disease spends its force on the serous coat, it is not unusual for the whole of the peritoneum to be studded with multiple tumours varying in size from that of a small shot to that of a hen's egg, each of which presents the characteristic microscopical features of the disease.

COLLOID CANCER commences most frequently in the serous or subserous tissues. Thence it extends by the lymphatic

vessels to the other coats, or it may also involve the whole of the peritoneum. The disease is principally characterised, not only by the thickening in the various coats which it effects, but also by the formation of various-sized blebs or vesicles, with walls of connective tissue, and containing a clear or blood-stained glairy fluid, which is discharged by ulceration into the peritoneal cavity or into the interior of the stomach.

Whatever the form of new growth be, its effects are both physiological and mechanical. It destroys the gastric glands in the area affected, and it produces obstruction to the passage of food. But the obstruction is not entirely mechanical. The calibre of the pylorus in health will only admit the tip of a little finger; and as in many cases which presented urgent vomiting during life it has been found, *post mortem*, that the pylorus, though involved, was even dilated, it would seem that the symptoms of obstruction were due rather to destruction of the muscular coat than to actual stenosis.

Symptoms.—Obscure at onset. *Dyspepsia* is the earliest symptom, attended by cachexia and progressive emaciation. These may occur for some months before the physical signs of tumour are present; but their presence in a patient over forty years of age should always make us suspicious. Then *Vomiting* supervenes, first at long intervals, then regularly after food. Its relation to food, as regards time, will vary according to the situation of the growth, being almost immediate if the affection be near the cardiac end of the stomach. According to Fagge, malignant disease may occur in the anterior wall of the stomach without vomiting. *Pain* is fairly constant, and is not necessarily increased by food. After a time, a tumour in the epigastrium may be detected on palpation. The abdominal walls over the tumour are resistant and rigid, and not unfrequently there is undue abdominal pulsation. As the disease progresses, the vomiting increases in urgency. The vomited matter is often intermixed with blood, which may be a bright red or 'coffee-ground' colour; it is extremely acid, owing to the presence of hydrochloric and other acids, and contains *sarcina ventriculi*. It is stated that the hydrochloric

acid is actually less in proportion than in health, but is replaced by other acids. When the pylorus is involved, it causes dilatation of the stomach, with eructations of large volumes of flatus, together with atrophy of the intestines. The bowels are confined, and the motions often black (melana) from admixture with blood. The progress of the disease is characterised by rapid emaciation, with mental despondency or irritability of temper.

Prognosis.—Always unfavourable. Death occurs in eighteen months or two years, generally from exhaustion, the senses being retained to the end.

Complications.—1. Rupture or perforation of stomach, and escape of contents into the peritoneal cavity. 2. Anasarca or ascites, from pressure of growth on the inferior cava or portal vein. 3. Jaundice, owing to secondary involvement of the liver or the bile ducts. 4. Fæcal fistula (with fæcal vomiting) between stomach and transverse colon.

Treatment.—Entirely palliative. The diet should be simple and digestible. As pain, however, is not so severe as in perforating ulcer of the stomach, a more extended dietary is allowable. Alcohol is not contra-indicated. Give opium if there be much pain or sleeplessness. For vomiting give ice, effervescent alkalies, or alkalies in combination with dilute hydrocyanic acid.¹ Flatulence may be controlled by ether, charcoal, or oil of cajuput. The bowels should be relieved by large enemata, to which olive oil may be added.

If the vomiting be excessive, and the vomited matter contain much sarcinæ, it may be stayed by washing out the stomach, or by the administration of dilute sulphurous acid. Nutrient enemata are frequently indicated. Excision of pylorus has been attempted, with some degree of success. It should, however, be performed early, or not at all.

DILATATION OF THE STOMACH

This condition may be partial or complete. *Partial* dilatation occurs in the cardiac end and first portions of the

¹ R. Sodii Bicarb : gr. xv. ; Acid: Hydrocyanici Dil: miiij. ; Potassii Iodidi, gr. v. ; Infusi Gentianæ Co : ad ʒj. (St. Thomas's Hospital.)

stomach, and is generally due to some form of new growth, or to cicatricial contraction of its walls, secondary to the healing of an ulcer or other abrasion of surface.

Complete dilatation is due to two main causes. 1. It may be due to pyloric obstruction, whether caused by cancerous growth, adhesion of pylorus to surrounding structures, or cicatrization of some ulcer. 2. It may also be directly caused by atony of the muscular walls of the stomach. This condition is produced by general enfeeblement, by chronic starvation, or by frequently overloading the stomach with large meals and an excessive amount of fluids.

Symptoms.—The disease is a chronic one. Cases of acute distension of the stomach have been recorded, but they are very rare. The principal symptoms are the ingestion of a large meal, vomiting afterwards at a period varying from a few minutes to many hours, bulging in the epigastric and left hypochondriac regions, and marked peristaltic movements of the stomach: all these being attended by severe discomfort and distress, and eventually succeeded by progressive emaciation.

The vomiting may occur almost immediately after food, or it may only take place during intervals of one or two days. The ejected matter, therefore, may be little changed; or its likeness to any food which has been taken is quite unrecognisable. The vomit which is produced at long intervals is of huge quantity, intensely sour and acid, probably offensive, and contains *sarcinae ventriculi*. The bulging corresponds somewhat to the shape of a distended stomach. It is, therefore, most pronounced on the left side of the abdomen; its lower and left-hand border, which may reach as low as the brim of the pelvis, can be ascertained by the tympanitic note afforded by percussion. Peristalsis is a remarkable phenomenon. It is visible to the observer as a wave in the anterior wall of the stomach passing from left to right, which causes a corresponding upheaval of the abdominal parietes, and which is elicited or excited by palpation of the epigastrium.

As may be imagined also, some of these signs may be aggravated by partially filling the stomach with some warm

water or other fluid, when also—in addition to the physical signs of its distension—the stomach yields, on shaking the patient, a musical splash or succussion note.

Discomfort is evidently due to pressure of the stomach on neighbouring organs. Emaciation may be more or less rapid, according to the amount of aliment actually absorbed. As a rule, however, the bowels themselves are empty and retracted, the stomach acting as a reservoir, and allowing very little digested food to pass the pylorus.

Treatment.—Galvanism to the epigastrium has been advocated. Strychnia may also be tried, with a view to stimulate the muscular coat and so cause contraction. These methods of treatment usually fail. The stomach should be washed out by passing into it a rubber tube with a funnel attached to the upper end. By this means medicated fluids can be poured into the stomach, and returned by holding the funnel end of the tube below the level of the patient's couch. The fluids used should be tepid water acidulated with hydrochloric or salicylic acid (one per cent.), or a solution of bicarbonate of soda (two per cent.). The stomach should previously be washed out by simple warm water.

The diet should be simple and digestible. Only a small amount of food should be allowed at each meal. In one case under our observation, potatoes dressed with milk was the only food for which the patient had any appetite or toleration.

DYSPEPSIA

Definition.—Imperfect digestion and assimilation of food ; or pain and distress during this process.

Causation.—(a) **PREDISPOSING CAUSES.**—*Age.*—It is most common at the extremes of life, but especially in old people when digestion fails from deficient gastric secretion, impaired muscular tone of stomach, and also weakened powers of mastication. *Climate.*—One is predisposed to the disorder by residing in a relaxing climate which reduces the vital powers. *Occupation.*—For similar reasons those suffer who follow sedentary or unhealthy callings, such as clerks, type-

setters, &c. *Heredity*.—It is frequently transmitted from parent to offspring. *Other Diseases*.—It may be secondary to phthisis or anæmia, by both of which the general tone of the system is lowered. It may be symptomatic of gouty inflammation of the stomach, or arrest of biliary secretions.

(b) *EXCITING CAUSES*.—*Food*.—Under this heading must be included imperfect mastication due to haste, defective teeth, or sore gums ; deficiency as well as excess of food ; meals which are imperfectly arranged as regards intervals, or as regards menu. The food may be improperly cooked ; or its constituents may be injudicious, such as salads, pickles, uncooked vegetables, jams, hard meats. An excess of alcohol, tea, coffee, and the abuse of tobacco, narcotics, and the like, will also excite an attack. Lastly, certain articles of diet (radish, curries) are peculiarly indigestible to various individuals.

Neuroses.—Such as a general deficiency of nerve tone ; mental excitability or strain ; nervous impulses reflected from disease of uterus, kidney, liver ; or excessive eye-strain and the like.

Vascular Derangements.—Such as passive congestions of the stomach secondary to chronic bronchitis, or valvular disease of the heart.

Abnormal Conditions and State of Health, due to the influence of cold, exposure, fevers, or to excessive muscular exertion.

Stomach and Duodenal Lesions.—These would include catarrhs, ulcers, malignant new growth, chronic dilatation, mechanical contractions, &c.

Functional Disorders.—It is notorious that in some individuals dyspepsia may arise from causes entirely misunderstood, and that with diet and surroundings which are apparently perfectly hygienic, they suffer from the disorder whilst others escape. These different conditions must be regarded as due to certain idiosyncrasies of the individual.

It is also obvious that many of the above exciting causes act together, and that they produce indigestion by some interference with the chemical process of assimilation of food, or

by disturbance in the muscular or mechanical arrangements of the stomach and bowels.

Those varieties in which inflammatory or other gross lesions exist may be grouped under the general term Chronic Gastric Catarrh, and the symptoms will be found described under the various diseases of the stomach.

The forms of dyspepsia which are not associated with congestion or other changes in the stomach or duodenum are known as Atonic Dyspepsia.

Atonic Dyspepsia

Symptoms.—It may occur at any age and in either sex. After eating there is a sense of weight and oppression at the stomach usually referred to a varying point behind the sternum or to the epigastrium ; but there is no pain and no tenderness. The distress is simply due to difficulty and slowness of digestion. It comes on about two hours after meals, and is followed by flatulent distension, the result of fermentation of ill-digested food. The patient has no appetite, or it disappears at the sight of food ; the tongue is usually clean, but flabby and indented ; the breath is heavy and foetid ; the bowels usually are constipated, although this may alternate with irritable diarrhoea from the presence in the intestines of only partially-digested food ; but the patient has neither vomiting nor hæmorrhage, nor can any epigastric tumour be detected. Consequent on the gastric distension he has palpitations, irregularities of heart, pains in the left side and præcordial region, and other symptoms which give rise to a fear of heart disease.

He complains of languor and sleeplessness, and yet, in spite of all these drawbacks, he does not emaciate as in organic disease, and there is complete absence of fever.

Treatment.—*Dietetic* most important. Food must be given in quality and in such quantity only as can be easily digested. Milk, jellies, strong soups, or meat extracts in small quantities at frequent intervals are best. This, however, applies only to the more severe cases. In ordinary forms the food must be of greater variety. Animal foods of all kinds must be freshly

cooked ; avoid warmed-up dishes ; mutton is more digestible than beef. White fish (sole, plaice), game, eggs may be allowed, and also certain vegetables such as potatoes (baked in their skins), spinach, French and haricot beans.

Certain articles of food, on the other hand, must be forbidden, such as cheese, pastry, new bread, jams, pickles, hard or salted meats, pork, and veal. Tea or coffee may be allowed if they agree, but should always be taken with much milk ; cocoa is probably preferable. Give three meals a day at intervals of five hours. A little alcoholic stimulant, such as claret or good whisky, may be taken at meals.

But we cannot make any estimate as to the digestibility of any article of food from its duration in the stomach, some dyspeptics having a peculiar aptitude for assimilating certain dishes which could not be taken by others. Also we must remember that the power of digestion is greatly influenced by the mind. Food which is appetising to one is disgusting to another ; and this is important, since the digestibility of a dish depends greatly on its taste and appearance, and according to the idiosyncrasy of the patient. For example, tripe and sweet-breads, which may be excellent articles of diet to some, become absolutely valueless to others, on account of the loathing which they produce. Our experience in obstinate cases is in favour of a prolonged fast for at least two days ; the patient meanwhile remaining in bed. By this means the stomach appears to recover strength by its physiological rest. In many instances, at least, the Catholic custom of fasting one day a week is attended by good results.

Supplement dietetic regimen by exercise, fresh air, change of climate, and attention to all the rules which should govern the pursuit of health.

Medicinal.—Drugs are only auxiliaries. To the anæmic dyspeptic give iron (syr. ferri iod.), or iron and quinine, to which aloes may be added if there is a tendency to constipation.¹ The vegetable bitters are also useful (calumba, gentian, quassia, nux vomica, and cinchona). In all cases an infusion or a

¹ R. Ferri Sulph: gr. ij.; Ext: Aloës Socot: gr. j.; Ext: Nucis Vom: gr. ss; Ext: Belladonnæ gr. ss. Fiat pilula.

decoction of the root or bark acts better than the corresponding dose of the active principle. Linseed in the form of a decoction or infusion has also a very soothing effect in flatulent dyspepsia. In most instances, however, it is advisable to frequently change the remedy.

Dyspepsia of Organic Disease (CHRONIC GASTRIC CATARRH)

Symptoms.—All the symptoms of atonic dyspepsia exist, together with others which are peculiar to the different lesions which originate the trouble. Space does not allow us to discuss separately the signs of each disease of which dyspepsia is a symptom, although some conditions, such as *Pyrosis*, *Gastralgia*, are so important as to require special description.

An attack of indigestion is frequently preceded by numbness or by formication in the limbs, and also, according to Herschell, by a sense of constriction and other unpleasant phenomena at the root of the nose, a symptom which we have frequently verified.

The attack itself is characterised by loss of appetite, or by an abnormal craving for food (boulimia), or by alteration in the sense of taste, by nausea, or by vomiting not always connected with food, by pyrosis, and by acid eructations. The vomited matter frequently contains *sarcine*. After food has been taken, there is epigastric pain and tenderness, a sense of burning, heat, or distress between the shoulders, the stomach is abnormally distended with gas, foul or acid belchings are frequent, the bowels are constipated or irregular in action. In addition, the patient suffers from palpitation, dyspnoea, and cardiac irregularity, owing to the mechanical disturbance of the heart caused by a distended stomach. He also suffers from certain nervous symptoms. He complains of drowsiness, headache, or vertigo, becomes mentally depressed or irritable; and he may, in chronic cases, lapse into confirmed hypochondriasis.

In dyspepsia due to duodenal disease, which may be secondary to gastric catarrh, or may be caused directly by

diet, the duodenal secretions are arrested, the intestinal peristalsis is increased, and diarrhœa results. This is usually brought about either by the too rapid onward flow of food which is excessively acid, the duodenal secretions thus being neutralised; or the food may be too rich in starchy and vegetable constituents, which produce a demand for pancreatic chemical action greater than it can afford.

Treatment.—In all cases it is imperative to observe the general rules for the maintenance of health. Exercise should be moderate and regular. The skin and other emunctories should present healthy actions. The clothing should be light and warm, and the patient must guard against chill and excessive heat.

The rules which guide us in the ordering of a dietary should embody these three essentials, viz. : (1) quantity of food should be only such as can be tolerated; (2) the quality of the food should be such as will pass muster as regards digestibility and preparation; (3) meals should be given at fixed times at equal intervals.

As regards medicines, the mineral acids appear to increase the alkaline and to diminish the acid secretions. On the other hand, bicarbonate of soda or of potash (grs. x. to xx.) before meals, aid digestion by increasing the gastric fluid. They also check fermentation and consequent flatulence, if given about two hours after a meal. Mineral acids acting in the opposite way should be given before meals in those cases in which dyspepsia is dependent on excessive gastric secretion. Nitro-hydrochloric acid is especially useful in the feeble digestion of children, or in adults who complain of offensive eructations accompanied by oxaluria and despondency.

Cannabis Indica is a true gastric sedative in neurotic dyspepsia accompanied by painful epigastric sensations.

Flatulence and distension of the stomach, which are dangerous in cases of feeble heart, should be treated by powdered charcoal or with strychnia. The latter gives tone to the muscular walls of the stomach, it increases the appetite, and clears the tongue and palate. It is especially useful in the dyspepsia of alcoholics, as it also tends to arrest craving for

drink. The tincture of *nux vomica* is more efficacious than the corresponding dose of *strychnia*. Creasote may be given to increase intestinal peristalsis, and as an alterative to the gastric secretions. In all catarrhal affections of the stomach *ipecacuanha* is valuable; small doses of opium may be added to allay nausea. Pepsin does not appear to be of much efficacy unless given in large doses.

For heartburn (*Cardialgia*) diminish the starchy elements of food. Relief will usually be afforded by alkalies with laxatives.¹ Occasionally alkalies fail; then give mineral acids, especially if there is a gouty history.

Gastralgia

By this term is meant pain at the stomach due to some local neuralgia. It is often combined with *cardialgia*. It is common amongst the poor, and in hysterical women. It may be the only symptom of stomach disease. Amongst other causes may be mentioned gout, mental anxiety or emotion, and the abuse of tea or alcohol.

The pain is severe, and is referred to the epigastrium, 'reaching through to between the shoulders.' It is described as a gnawing or burning pain, paroxysmal and irregular in its incidence, and lasting for a few minutes, or it may be for an hour or more. It is not increased by pressure, but is often relieved by it. It is apparently not related to food-taking, as it often supervenes some hours after a meal; and occasionally it is relieved by food, especially some of the less digestible kinds. Not unfrequently it is associated with flatulence and a rapid, palpitating, or irregular action of the heart. The bowels are almost always constipated. Between the attacks the digestion is either normal or remains in the same condition as is described under Atonic Dyspepsia.

Treatment.—The two main indications are to relieve pain and constipation. For pain, bismuth is the most useful drug.

¹ R. Magnes: Sulph: gr. xx.; Magnes: Carb: gr. xxx.; Tinct Cardam: Co: ʒi. xv.; Tinct: Zingib: ʒi. x.; Infus: Gentianæ Co: ad ʒj. Misce, ft. mist.

It may be combined with hyoscyamus. A saline purgative is necessary when the tongue is coated. Charcoal is useful in flatulence. If bismuth fails, give nitrate of silver, which acts as a direct sedative to the stomach. It should be given cautiously, and for a short time only.

Constipation is best met by simple laxatives combined with hydrocyanic acid as a sedative.¹

Pyrosis (WATERBRASH)

A symptom rather than a disease. The patient complains of pain or constriction at the stomach, followed by eructation of a thin, watery fluid, mostly alkaline, but which may be neutral or acid. It occurs generally in paroxysms, especially in the morning when the stomach is empty; it is increased by the erect posture and the regurgitation of gas. Women suffer more than men, and it has been attributed to poor food, or to a dietary consisting largely of starchy food (oatmeal, &c.). The source of the fluid is not yet determined; it may possibly emanate from the pancreas, or from the pyloric or salivary glands. There is a strong presumption that it is swallowed saliva.

Treatment.—Diet is the principal factor. Restrict farinaceous food. Meals should be taken at short intervals. If the symptom be severe it is generally relieved by sedative astringents (bismuth, kino, opium). Alkalies (sodii bicarb., potass. bicarb.) are indicated when the fluid is distinctly acid.

¹ R̄. Magnes: Carb: gr. xx.; Acid: Hydrocyanic: Dil: ℥iv.; Syrupi Rhei ℥xx.; Aquæ Pimentæ ad ʒj. Miscæ, ft. mist.

DISEASES OF THE BOWELS

ENTERITIS

Definition.—An inflammation of the intestines. The term is generally applied to inflammation of the mucous and sub-mucous coats. It is usually limited to the ileum (local enteritis), or it may extend to the jejunum, duodenum, and colon (general enteritis). The disease may be acute or chronic.

(a) Acute Enteritis

Causation.—The disease is more common in infants, during summer and autumn, and in warm climates. It may be caused directly by irritating food, medicines (drastic purges, acids), or it may occur as a local manifestation of cold (catarrh). It affects the duodenum frequently as a sequel to burns and scalds. Finally, it occurs in certain fevers either as a specific lesion, as in enteric fever and dysentery, or as a general febrile manifestation, as in scarlet fever and pneumonia.

Pathology.—The mucous membrane over the affected area is red and injected, and eventually becomes softened and thickened from inflammatory œdema. This condition extends to the submucous tissues; the follicles become enlarged, distended, and blocked with exudative inflammatory products; the cells lining the follicles undergo cloudy swelling, they proliferate, and finally degenerate into embryonic-like tissue-cells. This is succeeded by necrosis and ulceration. The ulceration may be limited to the follicles, or it may extend to a large area, so that wide tracts, or indeed the whole tube, may present the appearance of one large, ragged, sloughing surface.

Symptoms.—Vary according to the site and extent of the inflammation. Usually there is a somewhat febrile temperature (100° to 102°), a quickened pulse, with a sense of uneasiness in the belly. There are griping pains, most pronounced in the right iliac fossa, succeeded by diarrhœa especially when the ileum is affected. The stools vary in character, consisting first of undigested food, and subsequently of serous fluid and bile-stained mucus; when the rectum is involved the motions contain blood; they consist chiefly of mucus when the colon is the seat of disease; whilst if the affection extend to the duodenum they are freely mixed with green bile. In children tympanites is almost invariably present.

Diagnosis.—(i) FROM PERITONITIS: in which tenderness is very marked, the bowels are constipated, the pulse quicker and wiry. The affection may, however, extend to the peritoneum, when the symptoms of both conditions would be present.

(ii) FROM COLIC: in which there is no fever, the pain is spasmodic and more severe, and is often relieved by pressure.

Prognosis.—Favourable, as a rule; except after burns, and in children with a severe attack. The acute affection may pass on to the chronic, when the prospects of recovery are not so good.

Treatment.—Remove all causes of irritation from the intestinal canal by a gentle purge (castor oil best); excellent results follow the administration of calomel and ipecacuanha in children.¹ Diet should consist mainly of skimmed milk and farinaceous food. Benger's food is an admirable preparation. Fats and cream are contra-indicated, on account of their difficulty of digestion. Bismuth is the best medicinal remedy; it may be combined with opium when there is much pain. Watch the pulse, and give alcoholic stimulants (brandy) when there are signs of collapse.

(b) Chronic Enteritis

Causation.—It may supervene on an acute attack, or result from obstruction to the portal or general venous circulation. It appears to be endemic in hot climates.

¹ ℞. Hydrarg: Subchlor: gr. ij.; Pulv: Ipecac: gr. j. M. ft. pulv.

Pathology.—The intestinal follicles are enlarged, with their orifices patent and ulcerated. Their lining epithelium is either entirely lost, or represented by a few, straggling, fatty cells. The other coats, especially the submucous, are thickened by infiltration with embryonic cells. The surface of the gut is bathed in a thickish muco-pus, containing tube-casts and epithelium. There is also enlargement of the mesenteric and retro-peritoneal glands.

Symptoms.—Diarrhœa is excessive, accompanied by tormina and tenesmus. The stools contain mucus, or pus, with epithelium and epithelial tube-casts. The fever assumes a hectic type. The tongue is red and smooth, there is craving for food, yet progressive emaciation. The skin is shrivelled and muddy. In the advanced stages the mental condition becomes gloomy and hypochondriacal.

Treatment.—Diet is the principal thing. It should consist almost entirely of milk and farinaceous foods. If animal food is allowed, it should be in sparing quantities, such as weak beef essences, or fresh fish, or poultry. Salt meats, or hard, indigestible foods, and uncooked vegetables are especially unsuitable. The patient should guard against sudden chills by wearing flannel garments. The old-fashioned flannel roller cholera-belt round the abdomen affords relief and protection. Give an occasional mild purge of castor oil or calomel to clear the intestines. Diarrhœa may be controlled by sulphate of copper,¹ bismuth, or opium. Experience teaches that one of the best remedies is ipecacuanha in powder in large doses (gr. x. to gr. xxx.); a small quantity of opium may be added to stay its emetic property. When the colon is involved, excellent results are obtained by frequent large enemata of warm water, or of solution of nitrate of silver (from gr. $\frac{1}{8}$ to the ounce, gradually increased). Convalescence should be favoured by tonics and sea air.

¹ R. Cupric : Sulph : gr. $\frac{1}{2}$; Pulv : Opii gr. $\frac{1}{4}$; Pulv : Tragacanth : gr. iij. M. ft. pulv.

ULCERATION OF THE INTESTINES

Besides the specific ulcerations which occur in typhoid fever, tuberculous disease, syphilis and dysentery, the intestines are liable to other forms of ulcerous lesions in various parts of its course.

In the Duodenum ulcers form after severe scalds and burns of the skin. The pathology of this affection is at present obscure. The symptoms in the main resemble those of simple gastric ulcer. There is pain some little time after food—mostly situated in the right hypochondrium—vomiting, and melæna, and possibly some feverishness.

The Jejunum and Ileum are also liable to ulceration, not only in follicular enteritis, but as a result of pyæmia, pneumonia, and other febrile diseases. The symptoms are mostly ill-defined; but colic, diarrhœa, and hæmorrhage generally supervene at some period of the illness.

The Cæcum, with its appendix, and the large intestine generally, is similarly subject to ulceration. This condition is frequent in pneumonia, but it also occurs in chronic constipation. The ulcers are, for the most part, small, about the size of a sixpence; but, from the confluence of two or more, a larger lesion may result.

Between the ulcers the mucous membrane is swollen, congested, and perhaps blood-stained. Perforation of the bowel may result in chronic cases, and stricture after healing is not unknown.

The Rectum is also at times the subject of ulceration for a few inches above the internal sphincter, in fæcal accumulation.

Treatment consists in ordering light, non-irritating food, such as milk, eggs, and farinaceous puddings. Animal food is best avoided. Symptoms, such as colic and hæmorrhage, should be relieved by opium, turpentine, and other astringents. Our great object is to keep the bowels at rest as much as possible. In lesions of the large intestine the frequent use of bland enemata, so as to keep the mucous surface properly cleansed, gives the best results.

INTESTINAL OBSTRUCTION

Definition.—Mechanical impediment to the onward flow of the contents of the intestinal canal.

All cases, from a medical point of view, may be classified in two groups, viz. :

(a) Those in which the symptoms come on suddenly (Acute Obstruction).

(b) Those in which the symptoms come on gradually (Chronic Obstruction).¹

(a) Acute Obstruction

Causation.—1. **EXTERNAL HERNIA** (strangulated).—Detected by examining the various outlets, in belly-wall or pelvis, through which a hernia may protrude; always look for it, and examine for yourself.

2. **INTUSSUSCEPTION.**—The majority of patients are young and under seven years. The attack comes on suddenly with pain, which is often referred to the seat of obstruction, and vomiting, which usually, after three or four days, becomes faecal. Diarrhoea, with blood and mucus in the stools, is not infrequent at the beginning. A sausage-shaped tumour, formed by a length of gut invaginated into a portion below, may be felt through the abdominal parietes; it generally occurs in the ileum or caecum, and, as a rule, only one invagination occurs. About the eighth day, if the patient live as long, a slough of the middle (invaginated) tube comes away. Complete stoppage is rare, and at any time up to the date of complete occlusion by inflammation, there may be escape of flatus per anum.

3. **INTERNAL STRANGULATION.**—The usual conditions under this heading are volvulus, and strangulation, either by bands of lymph or fibrous tissue, or by congenital openings and malformations in the peritoneal cavity. It generally occurs in adult life. The symptoms come on suddenly, and consist of pain, faecal vomiting, and complete obstruction from the

¹ This classification is based on a clinical lecture by Murchison, delivered at St. Thomas's Hospital in 1878.

first, whilst the urine is scanty or suppressed. There is no external hernia to be found.

4. **IMPACTION OF GALL STONE.**—The majority of cases occur in women, and mostly in advanced life (over fifty-five). There is, as a rule, a previous history of biliary colic with or without jaundice, and of localised peritonitis denoting ulceration which allows the escape of the stone into the bowel. It must escape by this method, as the common bile duct would not allow the passage of a foreign body large enough to cause intestinal obstruction. The ulceration occurs invariably in the small intestine, and generally high up. The symptoms are characterised by sudden, intense, abdominal pain, incessant and marked vomiting, and scanty urine. The attack is rapid in its progress, whilst the symptoms may intermit in their severity on account of the stone moving.

5. **TYPHLITIS.**—The pain comes on suddenly in the region of the cæcum, over which there is circumscribed dulness and increased resistance. The vomiting is not frequent or urgent. There is often a history of a previous attack.

6. **EXTRAVASATION OF BLOOD INTO THE COATS OF THE BOWEL.**—Characterised by vomiting, with abdominal pain and tenderness where the obstruction occurs. Inquire for a history of previous hæmorrhage from the bowel or other mucous surface; for hæmorrhagic swellings on the surface of the body; or for a hæmorrhagic diathesis. It is a sufficiently rare condition to be easily overlooked.

(b) Chronic Obstruction

Causation.—1. **MALIGNANT NEW GROWTH.**—The patient is in advanced life, and has cancerous cachexia. There is a history of previous diarrhœa and hæmorrhage. The growth is almost invariably in the colon—especially its lower part—or in the rectum. The obstruction is slowly developed, and sometimes the tumour may be felt, if the abdomen be examined before the occurrence of distension and tympanites, which set in early. The patient may survive for weeks, or months even, after obstruction has taken place; the final collapse, however, is often markedly and unexpectedly rapid.

2. **SIMPLE STRICTURE.**—Characterised by absence of emaciation, cachexia, and tumour. It occurs most frequently in the large intestine. In most cases there is a previous history of dysentery and residence in tropical climates, or of syphilis. Examination of the rectum may reveal a stricture at a few inches from the anus. This is especially so in women, and is probably due to infiltration of the numerous fascial planes connecting the uterus and vagina to neighbouring parts.

3. **PRESSURE ON THE BOWEL FROM WITHOUT.**—Here there will be signs and outline of an abdominal tumour connected with some viscus, or evidence of enlargement of some organ, such as the liver or uterus.

4. **PARALYSIS OF BOWEL FROM SIMPLE FÆCAL ACCUMULATION.**—Owing to the accumulation, the bowel is often enormously distended, and loses its muscular tone. There is an absence of organic tumour; the patient is generally fat and weakly. The obstruction may be caused by the retention of certain foods (oatmeal, stringy vegetables), or by hair and foreign bodies which have been swallowed.

Symptoms.—There are certain symptoms which are common to all cases of obstruction from whatever cause, viz. :—

1. **IN THE EARLY STAGE.**—(a) *Distension*; (b) *Mapping out of, and vermicular movements in, coils of intestine*; (c) *Pain*, varying in intensity according to cause; (d) *Vomiting*, an early symptom if the obstruction be high up; (e) *Constipation*; (f) *Urine*, diminished, and sometimes almost suppressed. The quantity of urine depends on the seat of the obstruction, since the nearer it is to the stomach the more urgent the vomiting, and the less fluid, therefore, absorbed from the intestinal tract. When the small intestine is involved, all these symptoms are more severe than when the large gut is affected. Indican, also, is generally found in the urine in most cases.

2. **IN THE LATE STAGE.**—(a) *Paralysis* of movements of the bowel, with constant pain; (b) *Peritonitis and Enteritis*; (c) *General Collapse*.

Diagnosis.—FROM INTESTINAL COLIC.—In this condition the pain is more paroxysmal and spasmodic, with little or no

distension, and it is often relieved by pressure. The patient passes flatus. The trouble is relieved by simple measures, such as enemata, or mild purgatives.

FROM PERITONITIS AND PERFORATION.—There is usually some previous history of ulceration (typhoid, dysentery) or injury, &c., and not, as a rule, any history of constipation prior to the peritonitis. Further, the patient passes flatus.

Treatment.—Three principal objects are to be kept in view, viz.: (1) *Remove the obstruction if possible*; (2) *diminish peristalsis*, and so relieve pain; (3) *support the patient's strength*.

Search all the apertures through which a hernia may escape, and if found reduce the gut by means of hot baths, taxis, or operation.

INTUSSUSCEPTION.—Large enemata (air, warm water), opium, kneading the abdomen, or suspension of the patient head downwards, may have happy results. Laparotomy may have to be performed; but in this condition the symptoms are often not so urgent as in other causes of obstruction.

IN INTERNAL STRANGULATION, or VOLVULUS, give opium freely, and order large enemata. Then try the effect of abdominal taxis under chloroform, or of reversing the patient. If after this the symptoms continue and increase, an operation is imperative.

IMPACTED GALL STONE.—Medical relief can only consist in the free administration of opium and belladonna. Surgeons have succeeded in opening the abdomen and excising the calculus from the gut (Clutton, 'Trans. Clin. Soc.' vol. xx.), or in breaking it up by needle puncture without opening the bowel (Lawson Tait). If the symptoms remit, an operation is not advisable, as there is evidence that the gall stone is travelling.

TYPHILITIS.—The patient generally recovers if ordered complete rest in bed and given small doses of opium. The bowels should be relieved by oil-and-water enemata. Avoid purgatives, and order a scanty diet consisting of animal food rather than milk.

CANCEROUS AND OTHER STRICTURES.—Life may be pro-

longed and rendered more tolerable by surgical operation. Laparotomy, in these days of antiseptic surgery, is nearly always justifiable, except in cases of extreme age, feebleness, or collapse. If an operation be decided upon, the sooner it is done the better, as the sudden collapse which comes on twenty-four or forty-eight hours before death usually gives no warning of its advent.

In SIMPLE STRICTURE we must resort to iodides, mercurials, and anti-syphilitic remedies, as well as the mechanical treatment by bougies and enemata. A bougie may, however, be a dangerous instrument in unskilled hands.

In all cases the diet should be liquid and in small quantity; beef-tea or animal broths are best. Milk causes large, bulky evacuations, and is not, therefore, to be recommended. Iced water may be given, and also a little alcoholic stimulant, if required. Avoid purgatives, although the less violent drugs may be given in small quantities in faecal accumulation (castor oil, haust. sennæ co., calomel). Enemata are of the greatest service in nearly all cases, not only as a method of relief, but as a means of diagnosis. It is a good rule in all cases of obstruction in which the diagnosis is not clear to try their effect. They help to dilate a stricture, or relieve spasm; they not infrequently succeed in reducing an invaginated intestine; and they are especially useful in faecal accumulation. A good form of enema is to order half a pint of olive oil to be thrown into the rectum, to be followed by a pint and a half of soap and water slowly injected. As a rule, enemata are not to be recommended when symptoms of inflammation have set in.

Hot fomentations, bran poultices, &c., often afford much temporary relief; but the application of cold (ice bag or iced water) is better where there are inflammatory symptoms, when, also, leeches may be applied with advantage. In cases of great distension the gut may be punctured by small capillary trocar. Select those coils of intestine which seem most prominent and distended, with, of course, due anatomical precautions.

DIARRHŒA

Definition.—An excessive evacuation of loose motions.

Causation.—The direct and indirect causes of diarrhœa are numerous and most complex. Many of the conditions in the following categories may be due to more than one influence. We can only attempt, therefore, to group them approximately. The list, however, is by no means complete.

1. **FOOD** has an important influence. Diarrhœa may be caused by an excess of food, whilst, on the other hand, it may be due to deficiency of food, and it therefore occurs in starvation. Food again may be of an irritating character, or it may be insufficiently masticated, and so cause diarrhœa. Changes of diet, also, although perfectly nutritious and wholesome, frequently cause purging. This often occurs in children who have returned home from school with its plain, regular fare.

2. **CATARRH.**—In some subjects the ordinary malady known as ‘catching cold’ may manifest itself as catarrhal diarrhœa. It is possible, however, did we know sufficient of the causes of catarrh, that this variety of diarrhœa would be referred to the next category.

3. **POISONS.**—The diarrhœa occurring in the hot months of summer is probably due to some poison taken with the ingesta. It may also be readily caused by such poisonous food as decomposing fish and other animal matter. Impure drinking-water and offensive effluvia may act in a similar way. The effects of colocynth, elaterium, and other cathartic drugs need only be mentioned here.

4. **EXCESS OF INTESTINAL SECRETIONS.**—Diarrhœa supervenes on most of the conditions which cause an excessive overflow of intestinal mucus. Similarly the excessive flow of bile, which is due to the imbibition of large quantities of alcoholic liquors, notably of ale and stout, may be conveniently referred to here.

5. **NEUROSES.**—It is a well-known physiological fact that diarrhœa affects certain people under the influence of fear and terror, as also of joy, grief, and other mental emotions.

6. **FEVERS.**—Apart from the specific diarrhœa of cholera, dysentery, enteric and other specific fevers, febrility alone would appear to act as an exciting cause of diarrhœa. It occurs in the premonitory stages of measles and scarlet fever; also at the critical period of pneumonia. It is frequent in the many diverse conditions known as puerperal fever. The exact method of causation of the non-specific forms of diarrhœa is little understood. Possibly it may be due to weakened powers of digestion produced by the high bodily temperature; or it may be a natural manifestation by which the blood endeavours to rid itself of harmful matters which it contains.

7. **ULCERATION OF BOWELS.**—In all forms of ulceration of the mucous surface of the intestines diarrhœa is, at one or other period, a symptom—whether the lesion be tubercular, enteric, catarrhal, cancerous, or dysenteric.

8. **PERITONEAL LESIONS.**—Looseness or irregularity of the bowels is met with occasionally in peritonitis. Lardaceous disease of the intestines also, although not strictly a peritoneal lesion, may be here included as a cause of diarrhœa.

9. **PORTAL AND SPLENIC ENGORGEMENT.**—Similarly, any disease whatsoever which causes congestion of the portal system and its tributary veins, is accompanied by diarrhœa. The cause in these conditions is not the excessive secretion of bile, but hyperæmia of the whole intestinal tract.

Whatever the cause of diarrhœa may be, in every instance overstimulation of the peristaltic movements of the bowels is the exciting condition.

Symptoms.—Great latitude must be allowed in referring to the symptoms of diarrhœa. In a normal state of health one evacuation of the bowels should take place every day. Yet some people pass two or three stools a day without any departure from good health. In young children three or four motions a day is a normal condition.

Looseness of bowels with attendant pain and perhaps tenderness, are the principal symptoms. In many instances the diarrhœa is most severe at night, in others it occurs in the daytime only. Nevertheless, pain is not necessarily a constant

symptom. Colic and griping pains in the belly usually occur if the cause of the diarrhœa is in the small intestines only. Tenesmus, as a rule, points to the large intestine as the tract which is principally involved. Obviously the character of the stools varies considerably, according to the different causes of diarrhœa. Thus they may consist of mucus only, or of mucus mixed with blood, or of food which is partly digested, and therefore only slightly altered; they may be thin and watery in character, and in some conditions they show signs of putrefaction and fermentation, when they may contain *sarcinæ*, yeast fungus, or other products. In other cases they consist almost entirely of bile. These different characters of the evacuations have given rise to a nomenclature which, for the most part, is indicative not necessarily of any special disease or fever, but which is descriptive only of the appearances of the stools. Thus we speak of mucous, lenteric, choleraic, dysenteric, and bilious forms of diarrhœa.

In addition to the purging, other concomitant symptoms are usually present. The tongue is furred in the acute stages, but becomes abnormally red and dry as the purging becomes chronic. Vomiting is not infrequently present, especially in children. There is thirst, and there may be more or less fever, this latter symptom varying according to the cause of the diarrhœa. In the severe diarrhœa which often occurs in summer, other symptoms bearing a resemblance to Asiatic cholera may supervene, such as cramps in the belly and thighs, a coldness and blueness of the face and extremities, and other signs of collapse. But there would not appear to be any specific or contagious characters in this form.

Treatment will vary considerably, according to the cause of the diarrhœa. In children with symptoms pointing to irritation of the bowel by improper food, it is generally advisable to prescribe castor oil or grey powder, after which the purging often ceases. Compound rhubarb powder in such cases is also of great service. When the diarrhœa becomes excessive, some astringent medicine then becomes necessary. No drug is more useful than opium, which may be given in one or other of its many preparations. As regards the choice of mineral

acids and vegetable astringents, a good rule is to be guided by the condition of the patient's tongue. So long as it continues furred, vegetable astringents, such as catechu, kino, hamatoxylum, tannic and gallic acids, will generally be found efficacious, one or other of them. The aromatic powder of chalk is especially valuable in children.¹ The red and fissured tongue, on the other hand, is an indication for the exhibition of dilute sulphuric or nitric acid. The tincture of opium can be combined with advantage. That form of diarrhœa, especially seen in children, which is accompanied by offensive, watery stools will often yield to mj. doses of carbolic acid, which apparently cures by its antiseptic qualities.

In children, also, opium is not necessarily contra-indicated. One-drop doses of laudanum, or $\text{gr. } \frac{1}{50}$ of acetate of morphia, will frequently produce excellent results.

Diet, in all cases, is a very essential point. Hot drinks should be avoided. Green vegetables and fruits are also contra-indicated. Animal food, such as beef-tea, chicken broth, the expressed juice of raw steak, or even pounded steak itself, may be given. For children, milk should be boiled, and diluted with a third part of lime water. In exhausting diarrhœa, raw beef juice would at times appear to be the only treatment which will arrest it. Starchy, farinaceous puddings will also be found of service. Alcoholic stimulants may be required, the state of the pulse being the chief guide. In infants, a depressed fontanelle is an especial indication for alcohol. Give brandy in twenty-drop doses every hour.

CONSTIPATION

In normal health an adult should have at least one alvine discharge daily. Nevertheless, in numerous individuals an evacuation of the bowels occurs only every other day, or twice a week, or even at longer intervals, and with no departure from apparent health.

Causation.—Apart from structural disease of the ali-

¹ \mathcal{R} . Pulv: Cretæ Aromat: gr. iv. ; Sodæ Bicarb: gr. iv. ; Spt: Ammon: Aromat: mij. ; Aquæ Pimentæ ad ʒij. Misce.

mentary canal, which causes obstruction, the following list includes most of the causes of constipation.

AGE.—As a rule constipation occurs most frequently in elderly subjects, in whom bodily activity is less pronounced and the intestinal secretions less copious.

SEX.—It occurs much more frequently in women than in men, and especially in chlorotic girls.

HABIT.—It may supervene on laziness, indolence, and neglect of a natural demand to evacuate the bowels. This is a marked predisposing cause in young girls, in whom shyness may beget a constipated habit.

OCCUPATION.—Those who lead a sedentary life, and whose calling entails irregularity in habits and meals.

FOOD.—Certain foods produce constipation. In some the existence of fibres, husks, &c., produce accumulations over which the peristaltic movements of the bowel have no influence. Other foods by their easy digestibility leave little or no residue or fæces. Or, again, the fæces themselves may be preternaturally dry, or their moisture reabsorbed, as in diabetes. Indigestible articles of food also may in one person produce constipation, whilst in others diarrhœa results.

INTESTINAL DISORDERS.—Under this category must be included diminution or disorder of intestinal secretions, including bile, loss of muscular tone of the intestines, and, finally, painful diseases, such as fissure and excoriations, of the anus.

Symptoms.—The symptoms of constipation vary considerably in different individuals. In some people no discomfort whatever arises, and they are able to go about their usual work and daily routine without any sign of distress, even though an action of the bowels occurs only once a week. In most subjects, however, in whom a daily evacuation is almost a necessity, constipation produces well-defined symptoms. The patient suffers from frontal headache; the tongue is furred; there is a fœtid odour of breath; he complains that his mouth is clammy, and has a 'nasty taste;' his appetite fails; he has a sense of abdominal discomfort, and also of mental irritation and disinclination to follow his usual calling. As the condition becomes more chronic, the abdominal disten-

sion becomes more pronounced ; scybalous masses may possibly be felt in the course of the colon, or in the rectum ; the patient suffers from flatulency ; the colon becomes distended, often enormously ; he suffers from piles ; and, eventually, all the symptoms of intestinal obstruction from accumulation may supervene.

Treatment.—As a general rule strong purgative medicines are not required. They afford forcible relief for a time, and so long only as they are taken, but after their use the bowels tend to lapse into their previous state. The bowels should be coaxed into action, and not driven. This may at times be facilitated by a daily attempt at defæcation at a regular fixed hour, and by kneading the abdomen. Diet should be carefully regulated, and should include a plentiful allowance of green vegetables, oiled salads, fruits, and whole-meal bread. A sufficiency of fluids must also be taken. Regular daily exercise is also necessary.

If the above regimen is not sufficient to bring about the desired results, we must then resort to medicines. Laxatives had better be tried at first, such as small doses of castor oil, sulphate of magnesia, or one of the many saline aperient waters, such as Hunyadi János, or Friedrichshall. They are best taken in the morning whilst fasting, and their action is rendered more sure by a subsequent cup of warm tea, or a draught of warm water. In obstinate cases stronger cathartic remedies are required. Aloes, colocynth, podophyllin, cascara sagrada, and mercurials are, as a rule, safely tolerated. The griping action of some of these remedies can be obviated by the addition of hyoseyamus or belladonna. The rules for their prescription will depend on the peculiarity of the individual, and on the evidence which is afforded by inspection of the evacuations. Some cases respond to one drug, others require quite different medication. A good plan is to find by experience which aperient, or combination of aperients, produces the best results, and then to gradually diminish the dose until the bowels act spontaneously.¹ *Nux vomica*

¹ R. Ferri Sulph. ; Aloin ; Ext : Nucis Vom. ; Ext : Belladonnæ, āā gr. ss. Misce ; fiat pilula.

will also frequently keep up a healthy tone of the muscular fibres of the intestinal canal, and assist defæcation. Copious enemata of warm soap and water may be used with advantage when the accumulation in the lower bowel is excessive. They can do little or no harm even if continued indefinitely.

COLIC

Definition.—Abdominal pain occurring in paroxysms, and due to distension of bowel or of some duct.

For Biliary Colic, see p. 290.

For Renal Colic, see p. 556.

For Lead Colic, see ‘Plumbism,’ p. 482.

Causation.—Among the causes acting on the bowel are datus resulting from dyspepsia, improper food, sour wines and beers ; accumulation of and over-distension by fæces ; abnormal condition of the biliary and other secretions ; paralysis of the muscular coat ; the action of lead ; finally, it may be a manifestation of gout, or of catarrh affecting the intestinal canal.

Symptoms.—In intestinal colic the principal symptom is acute, agonising pain coming on in paroxysms, but with intervals of relief between. The pain is usually referred to the umbilicus, or to the situation of the transverse colon. It causes the patient to writhe about ; he is restless and cannot stay in bed ; his face is pallid, and often bathed in cold perspiration ; his pulse is quickened, but there is *no* fever. On examination, the belly is found to be distended and tympanitic, the tympanites being frequently irregularly distributed ; pressure often relieves the pain. The bowels are usually constipated, unless the colic be due to some irritant, when there may be diarrhœa.

Treatment.—Ascertain the cause of the trouble, our object being at once to relieve the intense pain. If it be due to indigestible food, it is speedily relieved by an emetic (mustard or ipecacuanha best). If the colon be distended, the enema of turpentine followed by pil. assafœtidæ comp. is effectual.

Opium or belladonna may be given in addition, for their

anodyne and antispasmodic effects. Subsequent attacks should be carefully guarded against by careful attention to the dietary. Meals should be small, and at intervals which are not too long. In elderly people it is of great importance to ascertain that their food is properly masticated, and not bolted in solid lumps.

ASCITES

Definition.—Dropsy of the peritoneal cavity.

Causation.—Divided into three primary groups : (1) obstruction to peripheral circulation in the peritoneum, from chronic peritonitis, tabes mesenterica, malignant new growth, tubercle, &c. ; (2) impediment to the circulation, occurring at the heart (valve-disease, fatty degeneration of walls), at the lungs (chronic bronchitis, emphysema), at the liver (cirrhosis, chronic congestion, lardaceous disease, tumours, in the transverse fissure), at the kidneys (Bright's disease) ; (3) disorders of the blood (anæmia, cancerous and syphilitic cachexia).

Symptoms and Physical Signs.—When fluid has collected to any appreciable amount, the belly becomes distended from below, this being most apparent when the patient stands up. Eventually the distension extends symmetrically to the whole of the abdominal cavity (unless the ascites be sacculated), so that the belly becomes smooth and rounded.

Percussion now detects areas of dulness over those parts to which the fluid gravitates, and high-pitched resonance where the intestines float to the surface. It is therefore obvious that these two signs, dulness and resonance, will vary in locality according to the position of the patient, whether lying on his side, or on his back, or standing. Although palpation affords a sense of tension and resistance, Dr. Ord has demonstrated the ease with which an enlarged or hardened liver may be felt, viz. by firmly placing the hand over the lower edge of that organ, then quickly pressing downwards, and thus suddenly displacing the fluid lying over its convex surface. The most important sign, however, is *thrill*. By placing one hand on the abdomen near the 'high-water' mark of the fluid, and

then tapping the belly-wall at the opposite side, a characteristic wave or thrill is distinctly felt. In addition, the fluid may give rise to certain pressure signs, such as œdema of the abdominal walls, distension of veins, protrusion of the umbilicus. The heart's action is feeble, and there is frequently dyspepsia. The serous fluid when examined after aspiration is of sp. gr. 1015, a greenish-yellow colour, of watery (not viscid) consistency, and contains much albumin.

Diagnosis.—(1) FROM OVARIAN DROPSY.—Here the enlargement is slow ; the tumour commences on one side in the region of an ovary, and is equally prominent on sitting down or standing ; dulness is pronounced towards the middle line, whilst the flanks are resonant ; there is no change in the percussion note on alteration of position. The fluid contained in the cyst is viscid.

(2) FROM PREGNANCY.—This condition would be diagnosed by the detection of foetal heart sounds and uterine murmur. Colostrum may be expressed from the nipples. The enlargement of the uterus is in the middle line.

(3) FROM TYMPANITES, in which there is absence of ascitic thrill, and a high-pitched drum-like percussion note all over the abdomen.

(4) FROM COLLOID CANCER OF THE PERITONEUM.—There would be evidence of prolonged emaciation and cachexia ; also irregular areæ of dulness and of resonance, and the fluid resulting from aspiration is thick and gelatinous.

Treatment.—No definite rules can be laid down, each case must be considered from its clinical aspect.

(1) If possible, REMOVE THE CAUSE OF THE ASCITES. Success in this, so far as the condition originates in heart or lung or kidney disease, can only be temporary. Still some advantage may be derived by the exhibition of digitalis in combination with squill and mercury.¹

(2) REMOVE THE FLUID by improving the condition of the blood with tonics, such as iron or the mineral acids. By

¹ R. Pulv : Digitalis gr. ss. ; Pil : Scillæ Co : gr. ij. : Pil : Hydrarg : gr. ij. Misce ; fiat pilula.

acting on the bowels with sulphates of magnesia and potash, pulv. jalapæ comp., elaterium (gr. $\frac{1}{2}$), podophyllum, colocynth, and other drugs which cause watery evacuations. They are best given in the early morning whilst fasting. By diaphoresis (hot baths, liquor ammoniæ acetatis). By diuresis (squill, digitalis, iodide of potassium). Occasionally cubebs and copaiba succeed when other drugs have failed. Finally by paracentesis abdominis. Do not delay the operation until discomfort or dyspnœa become urgent. In cases of cardiac disease the benefit is temporary only. In cirrhosis of the liver a good result is often of some permanency. It should therefore be performed early, and repeated if necessary. A tight abdominal binder applied after the operation will tend to prevent reaccumulation. Drugs, which failed to bring relief beforehand, may now be given, often with happier results.

(3) TREAT THE SYMPTOMS which may accompany, or be the result of, the ascites. Thus syncope or dyspnœa due to cardiac or pulmonary embarrassment point to the advisability of early paracentesis. Diarrhœa, independent of purgative remedies, may be an exhausting symptom, and will require special treatment.

PERITONITIS

Definition.—Inflammation of the peritoneum. It may be acute or chronic.

I. Acute Peritonitis

Causation.—(a) PREDISPOSING.—Age is important, as the disease is rare in children except at infancy from septic injury to the funis. It is also predisposed to by a previous attack, by the accumulation of fæces, and by debilitated constitutions.

(b) EXCITING.—*Traumatism*, such as blows and injuries. Under this heading may be included hernia, internal strangulation, intussusception, rupture of stomach or intestine, the bursting of abscesses or cysts, or of the Fallopian tube and escape of contents into the peritoneal cavity. *Various Ulcerative Perforations*, such as occur in typhoid fever, dysentery, typhlitis, and tubercular disease. *Extensions* of inflammation

from various organs and cavities (bladder, uterus, pleura, pericardium, intestine). *In association* with other maladies, *e.g.* Bright's disease, rheumatism, gout, scurvy. *Septic Fevers* (pyæmia, septicæmia, puerperal fever, erysipelas, small-pox).

Pathology.—The capillary vessels of the peritoneum are engorged, the membrane loses its smooth, shiny appearance, and becomes sticky from diminished secretion. The congestion is apparently most intense where the various coils are in apposition, hence they become glued together. This is followed by exudation of serous fluid, with more or less free lymph, tending to the formation of false membrane. As a result of the inflammatory process, the peritoneal coat also becomes thickened and pulpy, and is easily detached from the muscular tunic. Abscesses may form, and are usually circumscribed, especially in the omenta and in the pelvis. The affection may extend to the whole of the peritoneal surface, or it may be localised, for example, around the cæcum, or in the neighbourhood of the uterus or liver.

Symptoms.—Commence usually with a rigor followed by pain, at first limited to a certain area, and then extending to the entire abdomen. With this there are acute tenderness and distension, with resistance of the abdominal wall. The posture of the patient and his expression are almost diagnostic : he lies on his back, with legs drawn up to relieve tension ; his face is anxious, pinched, and the mouth drawn (facies Hippocratica). The pulse is quick (120 or more), sharp, and wiry. The temperature does not correspond to the intensity of the other symptoms. It usually ranges between 102° and 103° F., this comparatively low reading being probably due to shock. The skin is cold and clammy, the tongue is dry and coated, and the lips are often covered with sordes. The bowels are usually constipated, or there may be irregular diarrhœa. Nausea and vomiting are often urgent. The urine is of febrile character ; strangury may be present when the bladder is involved, and it is frequently accompanied, in children, by frequent urination, and pain down the thighs.

Death is generally due to collapse, the mind being clear to the last, though occasionally there are convulsions and coma.

Diagnosis.—FROM COLIC.—Here there is no fever; the pulse is full, the belly is not tender, and the pain is frequently relieved by pressure. FROM Hysteria.—The pain and tenderness are apparently so intense that the patient complains as soon as the hand is placed on the abdomen. Engage the patient's attention, when manipulation can be easily carried on. In addition there is an absence of distressed countenance and of fever. FROM ENTERITIS.—Here the vomiting is nearly always urgent; there is diarrhœa all through; the pain and tenderness are not so evident; and the pulse is softer and fuller.

Prognosis.—Always a grave disease. Yet recovery is to be anticipated, except in intestinal perforation, in severe injury, or in debilitated subjects. Favourable symptoms are the subsidence of pain, tenderness, and distension; cessation of vomiting; a slower pulse; and a less anxious expression of face. An unfavourable termination is to be looked for when there is an aggravation of symptoms; when the pulse increases in frequency and is less in volume; when pain diminishes without corresponding amelioration of other symptoms; when the extremities become cold, with a sudden fall of temperature, a shrunken countenance, and other signs of collapse.

The disease may run into the chronic form.

Treatment.—First ascertain the cause, as this will materially affect procedure. In general, however, the best results are obtained by keeping the patient under the full influence of opium. This may be done by giving the drug by the mouth in combination with calomel, by enemata, by hypodermic injection of morphia, or by suppository. The peristaltic movements are thus lessened and pain is diminished. A cradle should be placed over the abdomen. In strong, healthy subjects, and in cases due to mechanical injury, we may apply leeches (eight to twelve) to the abdominal parietes. Other external applications are ice, hot fomentations and poultices, and opium or belladonna plaisters. Distressful distension may be relieved by assafœtida, or by turpentine, either as stupe or enema (best), or by the mouth if vomiting be not severe.¹

¹ ℞. Ol: Terebinth: m.v.; Mucil: Tragacanth: ʒj.; Aquæ Menth: Pip: ad ʒj. Misc; ft. mist.

Some physicians have recommended constant purgation (sulphate of magnesia), whilst good results have been claimed for laparotomy and irrigation of the peritoneal cavity.

II. Chronic Peritonitis

Causation.—(a) **PREDISPOSING.**—1. A previous acute attack. 2. Some general disease in which the blood is altered in character (rheumatism, Bright's disease).

(b) **EXCITING.**—1. Inflammation of any of the abdominal organs, especially of the stomach, liver, bowel, and uterus. The conditions of these viscera, which give rise to chronic peritonitis, may be of simple inflammatory nature; or they may be involved in cancer, tubercle, or other new growth; or they may be subject to injury, parasitic invasion, &c. 2. New growth primarily affecting the peritoneum, *e.g.* cancers tubercle. 3. The effects of injury, amongst which we include frequent paracentesis abdominis, chronic inflammation of an old hernial sac, resulting from dragging or constant manipulation, injuries from prolonged pressure, as in the use of mechanical tools.

Pathology.—On examining the abdominal cavity there will be found thickening of the peritoneum, with adhesions between the various intestinal coils or between the opposing surfaces of viscera. The thickenings may be general, involving the serous and subserous coats of the intestines and viscera, or they may be local only, and confined to one region, or to one organ and its neighbouring intestine. Similarly the adhesions may be entirely local, fastening, *e.g.*, the uterus to the rectum and small intestine, or cæcum to the parietal peritoneum; on the other hand, they may be general, when the whole of the abdominal organs, intestines, and omenta are agglutinated together in one conglomerate mass in which the various constituents are with difficulty recognised. One of the effects of local chronic peritonitis is the formation of fibrous bands or bridges, causing danger from incarceration or strangulation of gut.

In addition to the above, the belly cavity will contain the fluid results of inflammation. That is to say, there will be

serum or pus. The serum may either be clear or turbid, or contain flakes, or be bloodstained: its quantity and the different characters will, in a great measure, depend on the extent and the duration of the inflammatory process.

Signs and Symptoms.—The abdomen is enlarged, but not always symmetrically, especially if the inflammation be local only. A percussion thrill may be usually obtained all over the abdomen, but occasionally only here and there. Local coils of intestine which are pushed to the surface or held there by adhesions will give a characteristic resonant note. In advanced cases, however, these signs may be absent, and instead, we find an irregular, resistant, abdominal wall, with a boggy or crepitant feel in different parts. The respiration is thoracic, not owing to pain, as in acute peritonitis, but from fixity of the diaphragm and abdominal organs.

The effects of the inflammation on the system generally are an increased temperature (100° to 101°) with exacerbations from time to time, abdominal pain of a dull or sub-acute character, alternating with attacks of colic, together with a sense of tightness and dragging, which is severer at one time than at another. The effects on the different abdominal organs are mainly those of contraction and pressure. Thus we may have symptoms of atrophy of spleen and liver, contraction of stomach, jaundice from obstruction, œdema of lower limbs, internal strangulation or twist of gut, and the like. The bowels are usually confined; they are not infrequently, however, irregular in action, with alternating attacks of diarrhœa and constipation. Vomiting occurs from time to time, and the urine is generally scanty from the impeded intestinal absorption. As the disease progresses, the patient becomes anæmic, feeble, and emaciated; the exhaustion gradually increases; food is refused, or if taken, with difficulty retained; aphthæ form on the tongue and inside the cheeks; and he gradually sinks from asthenia. The fatal termination is hastened by suppuration of the peritoneum, with discharge of pus by the bowel, bladder, uterus, vagina, or other outlet.

Prognosis.—That form secondary to cancer is invariably

fatal. The formation and discharge of pus also renders the prognosis more unfavourable. In tubercular peritonitis the prognosis is more favourable, especially in children. And in the ordinary forms of chronic peritonitis the outlook is also favourable ; but hope diminishes with recurring attacks.

Diagnosis.—The diagnosis of the condition is not so difficult as that of causation. We generally find a history of a previous acute attack, or signs of new growth in connection with some abdominal or pelvic organ. In tubercular peritonitis there is generally an excess of fluid. We are further guided by the appearance of the patient, the family history, the presence of pleurisy with effusion, of signs of ulcer of the bowel, or other evidence of tuberculosis. Aspiration of the abdominal fluid will help to elucidate the diagnosis.

Treatment.—If the condition be secondary to new growth our treatment can only be palliative. In tubercular peritonitis, iodine and cod-liver oil are indicated. We may give iodide of potassium internally. Children tolerate it when given in small doses (gr. ij.) in milk. We can also apply iodine paint or ointment to the abdominal walls. The syrup of the iodide of iron is also an admirable remedy. In simple chronic peritonitis we should promote absorption of the fluid by application of oleates of mercury to the abdominal wall. Firm, equable pressure of a flannel roller or an elastic abdominal belt also assists. We may, in addition, promote free diuresis by squill, the potash salts, liq. ammoniæ acet., &c. Finally, we may resort to paracentesis.

It is also important to keep the bowels freely and regularly relieved (calomel with opium), and to attend to flatulent and dyspeptic troubles.

TYPHLITIS AND PERITYPHLITIS

Definition.—Inflammation of the cæcum and surrounding tissues. The disease may originate in the cæcum ; or in the appendix.

Causation. — **PREDISPOSING.** — *Age.*—More frequent in adults. *Sex.* — Men suffer more than women. The disease tends to recur in those who have had a *previous attack*.

EXCITING.—Faecal accumulation ; faecal concretions (which usually consist of earthy phosphates, enteric mucus, and faecal matter) ; irritant foods, especially such as contain seeds, stringy fibre, husks, or small bones ; a large meal after a long fast. It may be brought on also by direct blows and injuries ; by strains, as in lifting. It may result from extension of the inflammation attending ulcers in the caecum (typhoid, dysenteric) ; or, finally, it may be due to abscess in connection with the ileum, or with the ovary, extending to the caecum.

Pathology.—It is probable that most cases of typhlitis from accumulation commence in the appendix vermiformis ; whilst those which are due to ulceration have their origin in the mucous surface of the caecum. Whatever be the cause, the mucous membrane becomes congested and swollen, the epithelium proliferates, the mucous glands pour out an increased secretion, and there is thickening of the submucous coat. At this point the inflammatory changes may subside and the results become absorbed. On the other hand, the inflammation may extend to the subserous and serous tissues, causing a local peritonitis, with adhesion of the caecum to the abdominal parietes and local viscera ; or the peritonitis may be diffuse, extending across the middle line and upwards to the whole of the peritoneal surface. The formation of pus which ensues may consequently be localised to the caecum by reason of adhesions, or the peritoneal cavity may practically become one large abscess. Subsequent to the inflammation of the mucous surface is the formation of one or more ulcers which may perforate the caecum with large or small openings, setting up perityphlitis and abscess. The abscess may point and be discharged through the abdominal wall, or by the inguinal canal, or through the various apertures in the pelvis, or by the various channels of the pelvic viscera.

Symptoms.—Generally commence with a rigor, followed by pain and discomfort in the right iliac fossa. Examination then reveals, at first resistance and dulness, subsequently a distinct localised tumour which varies in size according to the distension of the caecum and the extent of the inflammatory deposit. The tongue is coated ; there is either obstinate constipation as in obstruction, or an irregular diarrhoea. As a result of pressure

on the nerve trunks and large veins of the locality, the patient complains of pains extending to the lumbar region or down the thighs, and of œdema of the lower limb. The thigh, as a rule, is not flexed on the abdomen as in acute peritonitis. Under appropriate treatment these symptoms may subside, or the inflammation may terminate in abscess, when the tumour becomes more acutely tender and has a boggy or crackling feel on palpation. If it evacuate itself by external sinus the discharge consists of pus, containing gas, feculent matter, and perhaps seeds or other foreign substances. The case may terminate in death from general peritonitis, or from exhaustion; or in recovery after a prolonged illness.

Treatment.—The majority of cases recover when treated by complete rest in bed, a scanty, liquid diet, and the exhibition of opium. Usually nothing else is required. Avoid purgatives. Treat constipation by large enemata. The local pain and inflammation may be met by hot fomentations and poultices. If the condition go on to suppuration, the sooner the pus is evacuated the better. The evacuation of an abscess in connection with the cæcum, and the removal of the appendix vermiformis, are triumphs of modern surgery. But we should sanction removal of the appendix, only after two or more attacks of typhlitis.

ABDOMINAL TUBERCULOSIS

Definition.—Tuberculous disease of the intestines, of the peritoneum, or of the mesenteric glands.

Causation.—Although the respiratory tract is the most frequent site of primary tubercular affections, it is by no means always so. The abdominal cavity may be involved primarily or secondarily; and, indeed, it may be the only local seat of tubercular disease. There are no special causes—beyond those spoken of under Phthisis—why the abdomen and its contents should be the seat of tuberculous deposit. Possibly the intestinal mucous membrane may be directly inoculated by the milk or flesh of tuberculous animals; and this may occur especially in children. In

adults, the ulceration of the small intestine is generally due to swallowing bacillary sputa.

Pathology.—In the mucous membrane of the bowel the lymphoid tissues of the solitary and agminated glands are the seats of tubercular deposits. These glands first enlarge and then ulcerate; the ulcerous lesions, studded with tubercles, then become foci of infection, from which the disease extends centrifugally. The ulcers are thus at first separate, with zones or areas of healthy tissue intervening; but as the ulceration extends by the agency of the blood-vessels, neighbouring lesions meet and coalesce, the erosion tending to extend transversely round the gut until a complete ulcerous girdle is formed. The large and small intestine, with the intervening cæcum and ilio-cæcal valve, are all equally liable to be involved, owing to the universal occurrence of solitary glands in all these parts; but the ileum always appears more extensively infected, this being accounted for by the presence of Peyer's patches. Nevertheless, tubercular deposits have been known to stop short of ulceration, and, in rare cases, to heal after ulceration has occurred, whereby cicatricial deformities and even intestinal obstructions have ensued.

Peritoneal tubercle may be found scattered all over the serous sac, under the diaphragm, in the mesentery and omenta, and in the serous coats of the various organs.

In acute tuberculosis the nodules are of the discrete grey variety. In chronic cases they are found as large, yellow masses which have undergone, or are undergoing, all the caseating, retrogressive changes which are common to tubercle. In the majority of cases, however, the patient has succumbed before this stage can have arrived. Two widely different conditions may, therefore, occur. In the one, the discrete tubercle is associated with serous effusion, which constitutes a form of ascites. In the other, the tubercular growth, being more chronic, causes adhesion of the intestinal coils to each other, and possibly local accumulations of pus, and even fistulous communication between neighbouring portions of gut. In this latter variety the great omentum is frequently contracted and nodular, the nodules resulting partly from

contractions in this fold, and partly from agglomerated masses of tubercle, which may be felt through the abdominal wall.

The lymph glands may also be involved, but usually this is secondary to tuberculous disease of the serous membrane or of the mucous surface of the intestines. The glands enlarge, undergo caseation, and possibly suppuration. Their presence, also, may be detected by palpation of the abdomen in thin, spare subjects.

Symptoms.—Although one would expect to find pain and tenderness of the abdomen, as in idiopathic forms of chronic peritonitis, these symptoms are oftener absent than present. There is hectic fever always; and the belly is usually distended, and resistant in parts where there are large tuberculous accumulations, whilst in others it presents a boggy or doughy feeling, as though the intestines were matted together. Ascites may occur in one patient, as described above; in another it may be absent. Occasionally, however, the abdomen is retracted or ‘boat-shaped,’ the skin covering its walls being harsh and furfuraceous. Absence of abdominal breathing is always a marked sign in this, as in all other forms of peritonitis.

If the bowel itself be ulcerated, diarrhœa and hæmorrhage occur at some period or other of the illness; but diarrhœa may exist without tubercular enteritis, and *vice versa*. One frequently supposes that diarrhœa is indicative of ulceration, when it is merely a manifestation of hectic fever, as in pyæmia. The evacuations are generally yellow-coloured and most offensive.

Beyond the above, we find loss of appetite, loss of flesh, and diminution of bodily strength. Other symptoms of tubercular disease of the belly are common with those met with in chronic enteritis and peritonitis.

Diagnosis.—The salient points in the diagnosis are not difficult, so far as the detection of peritonitis is concerned. The tubercular element is the one which causes some difficulty in coming to a conclusion. We should, however, be guided by the presence of more or less distinct tumours in the abdominal cavity, the youth of the patient, the presence of

hectic fever, and a previous history of tubercular invasion of the lungs, or, indeed, the active presence of tubercle in these or in other organs.

Prognosis.—As a rule, unfavourable, especially in adults. In children the outlook is not so gloomy. In them, tubercular peritonitis and enteritis frequently tend to cure. Cases often occur in which abdominal tubercle undoubtedly existed, but of which no trace can be found, say, in twelve months after, especially if the child be regularly treated with cod-liver oil and placed amid favourable hygienic surroundings.

Treatment.—As in pulmonary phthisis ; the main indications are to attend to the general health, to improve the appetite so that wasting tendencies are checked, and to order a long-continued course of cod-liver oil.

All complications, such as diarrhœa and hæmorrhage, must be treated on general principles. Opium is of especial value, not only for checking diarrhœa, but also to relieve pain. Oleates of mercury have been recommended as inunctions to the abdominal walls.

For peritoneal tuberculosis, laparotomy, of recent years, has been ably advocated and successfully performed with good results by Mr. Howard Marsh and other surgeons, the tubercle bacillus apparently perishing as a result of the operation, by which purulent and serous collections are evacuated and the abdominal cavity thoroughly cleansed.

ABDOMINAL TUMOURS

Multiple and various tumours may exist in the abdomen. They may arise in connection with any of the different organs of the belly, as well as from its membranes and vessels. They include not only new growths, but dilatations of blood-vessels and tubes, accumulations of various secretions and excretions, parasitic cysts, and malformations. To treat each subject exhaustively would require space beyond the dimensions of this work. We can, therefore, only point out some of the principal tumours, with their most typical signs.

A tumour or growth originating in any one of the nine

regions of the abdomen obviously suggests its origin with one or other of the organs or structures which are situated in that region. It is necessary, therefore, to inquire into its history, the region from which it originates, its previous duration, the direction of its growth, and then to examine the tumour by palpation, percussion, auscultation, mensuration, and possibly by surgical exploration.

STOMACH.—This viscus may be so enormously distended as to almost fill the abdominal cavity, and even displace some of the thoracic viscera. In moderate distension the epigastrium, which is concave in typical health, is bulged forwards. The viscus yields a tympanitic percussion note over the front, by which its outlines can be easily mapped out. Owing to its containing more or less fluid, some alterations in the position of dulness may be detected as the body is turned from one side to the other. For the physical signs of malignant new growth, see Malignant Disease of the Stomach.

INTESTINES.—Distension of the bowels may be due to paralysis or to atony of the muscular fibres, or to the various forms of intestinal obstruction. It is also seen in enteric fever and in hysteria. The distension may be general or local.

LIVER.—Enlargements of this organ may be limited to one or other lobe, or they may be universal. Amongst the most frequent causes of general enlargement are amyloid degeneration and the early stages of cirrhosis. Local enlargements may be caused also by cirrhosis, but more frequently they are due to hepatic abscess, to hydatid cyst, or to distension of the gall bladder. We are led to a diagnosis by a smooth condition of the surface of the tumour (suggesting amyloid disease), or by its nodular character in syphilis, cancer, and cirrhosis. A thrill is confidently described as being present in hydatid cyst, but this sign is by no means easy to elicit, and in our experience it is not reliable.

In the history of any liver-tumour the growth generally extends downwards from the right hypochondriac or epigastric regions.

The **SPLEEN** may be enormously enlarged in chronic ague, leucocythæmia, and amyloid degeneration. As the organ

increases in size it appears below the costal arch, and extends downwards and forwards. It is recognised by its smoothness, firmness, and the notch in its anterior border; it also rises and falls with the diaphragm. It is only when the splenic hypertrophy is so exaggerated as to almost fill the entire abdominal cavity that it becomes difficult to diagnose.

KIDNEYS.—Tumours in relation with these organs are new growth, hydro- or pyo-nephrosis, cystic disease, perinephric abscess, hydatid cyst, and movable kidney. In examination for any of the above conditions the patient should be placed prone on his face, and the kidney dulness mapped out in the loin from the overlapping colon. The outer or anterior border of the kidney is about four fingers' breadth from the spinous processes. Examination from the front is also necessary. The kidney can often be felt in meagre subjects; but palpation is always more effectually performed by making the patient lie on the flat of one's hand, by which the organ can then be pushed forward and explored by the other hand. Occasionally, especially in floating kidney, examination in the knee-elbow position affords valuable evidence. But the existence of small or even moderate-sized tumours can rarely be detected by manipulation. The evidence obtained by the urine outweighs all digital examinations.

The **PANCREAS** also cannot be felt except in emaciated people. Cystic tumours, whether hydatid or retention, may exist. Malignant tumours are also not unknown, but comparatively rare. They are usually attended by jaundice, ascites and fat in the motions. Pancreatic tumours are fixed—that is to say, they do not share in the movements of respiration, although they may generally, by manipulation, be easily moved about, and even made to cross the middle line of the backbone. Pulsation from the aorta or some of its large trunks is generally more or less imparted. Stomach resonance can nearly always be obtained anteriorly to the tumour.

UTERUS AND OVARIES.—Uterine tumours are, for the most part, known by their density. Their relation with the uterus is detected by the use of the uterine sound, by changes in the

length and position of the uterus, and by the existence of metrorrhagia.

Ovarian cysts have a tendency to extend laterally, although they are frequently median in their position. Thrill may be usually obtained by percussion. The flanks are, as a rule, resonant, and there is no alteration in the position of the dulness on change of the patient's condition, thus pointing out that the fluid is incarcerated.

BLADDER.—Urinary retention may cause dulness and other signs of tumour, as high as the umbilicus, or even higher. The enlargement is always median in position and globular in shape. In case of doubt, a catheter will set the matter at rest.

PHANTOM TUMOURS occur in hysterical women. They are median in position usually, globular in shape, and are limited by a constricting zone above and below. But they may exist in any part of the abdomen and assume any shape. They disappear under chloroform.

DISEASES OF THE LIVER

ANATOMY

The **Liver** weighs about fifty ounces or more (three to four pounds).

It occupies the right hypochondriac, the epigastric, and slightly the left hypochondriac regions. The upper or convex surface fits into the dome of the diaphragm, and extends upwards to the following landmarks, viz. :—

In the vertical line of nipple to the fifth intercostal space.

“ “ “ mid-axilla to the seventh intercostal space.

“ “ “ inferior angle of scapula to the ninth intercostal space.

Its lower margin, posteriorly, is at the eleventh intercostal space.

The anterior margin of the liver is thin and sharp. It follows the costal arch in the male, and stretches across the epigastrium, from the ninth right to the eighth left costal cartilage. In women it usually projects about an inch below this. The gall bladder projects from this border, and impinges against the cartilage of the ninth rib, on the outer border of the rectus muscle.

The posterior border lies on the backbone, the pillars of the diaphragm, and the aorta and inferior cava.

The under surface covers part of the stomach and the duodenum, and is also in contact with the hepatic flexure of the colon, the right kidney, and suprarenal capsule. It is not necessary here to describe the fissures and their contents, except to remind the reader of the existence of one or two lymphatic glands in the transverse fissure.

SIMPLE ATROPHY

Definition.—A diminution in size and weight of the liver, without any alteration in its histological characters.

Causation.—*Advanced age.* *Inanition*, from mal-assimilation, from malignant disease of stomach, or from defective supply of food. *Pressure* on the organ by tight-lacing. *Effusions* in pericardium, pleura, or peritoneum. *Perihepatitis*, and pressures exerted by enlarged neighbouring viscera.

Pathology.—The organ is smooth and diminished in weight. Its lobules are wasted, but the cellular structure is by no means entirely wanting.

Symptoms.—No special symptoms beyond diminished liver dulness; other symptoms masked by the more important preceding illness.

Treatment.—Secondary to relieving the cause.

CONGESTION

Definition.—Hyperæmia of the liver, whether as a consequence of increased supply of blood to, or impediment to the return of blood from, that organ.

Causation.—Three groups: 1. ACTIVE; 2. PASSIVE; 3. MECHANICAL.

1. ACTIVE CONGESTION is found in weak, indolent persons leading sedentary lives, who are given to over-eating, especially highly-seasoned foods, and who consume an excess of alcohol. Common in tropical climates. It may occur as a result of malarial, yellow, relapsing, and other fevers. It is a frequent condition in inherited syphilis. Lastly, it may result from direct injury.

2. PASSIVE CONGESTION.—According to Murchison, this form may be the result of habitual constipation, insufficient exercise, or the arrest of any habitual flux (catamenia, hemorrhoids, &c.).

3. MECHANICAL CONGESTION is the most common form of hepatic congestion. Any condition of lungs or of heart—whether of walls, valves, or trunk vessels—which disturbs the

onward flow of the blood, will eventually cause congestion of the liver. Thus, in anatomical order, it may occur as a result of aortic obstruction, aortic regurgitation, enfeebled ventricular walls, mitral stenosis or regurgitation, chronic disease of lungs (bronchitis, phthisis, asthma, &c.), stenosis of pulmonary artery, insufficiency of pulmonic semilunar valves (rare), tricuspid valve disease, aneurysm or other tumour pressing on the inferior cava, &c.

Pathology.—The organ is enlarged, and its anterior edge slightly rounded. It is full of blood. The lobules are distinct, each having a red, congested centre with pale periphery—in other words, the central hepatic veins are engorged, whilst the circumferential portal vessels are free. The term ‘nutmeg liver’ has thus been applied, from the resemblance a section of the organ bears to a cut surface of a nutmeg. The microscope shows great engorgement of vessels in the centre of lobules. Blood cells are here crowded together, and compressing the liver cells, cause their atrophy. Pigment is often deposited in the liver cells and intercellular matrix. Fatty infiltration occurs at the circumference.

Symptoms.—The more marked symptoms occur in the active form of congestion. They are generally such as point to disordered liver-digestion, viz. nausea, flatulence, distension of stomach, enlargement of liver without pain, and, at times, jaundice. The splenic dulness is often increased as a result of the circulatory obstruction in the liver, and hæmatemesis also occasionally occurs. The urine is loaded with lithates, and glycosuria is by no means uncommon. (Ord.)

Treatment.—In the active form, a strictly regulated diet is essential. Give easily-digested, plain, and non-irritating food. No alcohol whatever. Saline purgatives, sulphate of potash, soda, or Friedrichshall or Hunyadi János waters may be given in warm water before breakfast. Chloride of ammonium is highly praised by Murchison,¹ and so is ipecacuanha. They may be prescribed with cascara sagrada or other active aperient. Afterwards give iron tonics with purgatives.

¹ R. Ammon: Chloridi gr. vij.; Ext: Cascaræ Sagradæ Liq: mx.; Aquæ Menth: Pip: ad ʒj. Misc.

In the passive form, secondary to some condition or disease of heart or lungs, treatment is directed to the primary cause. Even here much direct good may be done by leeches over the liver, together with small doses of blue pill.

ACUTE YELLOW ATROPHY

Definition.—An acute atrophy of liver, occurring mostly in pregnant women, attended by non-obstructive jaundice, cerebral symptoms, and the ‘typhoid state.’

Causation.—(a) **PREDISPOSING.**—*Age and Sex.*—Mostly in young adults; females 70 per cent. of cases. *Pregnancy.* *Dissipation.* *Spirit-drinking.* *Syphilis.*

(b) **EXCITING.**—*Nervous Influences*, such as grief, fear, joy. *Malarial Poisons.* *Specific Fevers*, notably typhus. *Phosphorus Poisoning.*

Pathology.—The liver is diminished in size and weight, and is flabby and puckered. The cells are atrophied, or entirely disappear; a few nuclei are seen here and there. In place of the cells there is fat, or pigment and granular matter. Leucin and tyrosin are found in the hepatic tissue except in phosphorous poisoning. Hæmorrhage of a petechial character occurs in the submucous tissues, serous membranes, and various organs.

Symptoms.—Slight premonitory signs, chiefly of disturbance of gastro-intestinal tract. Jaundice is usually the first symptom noticed. The early part of an attack is generally marked by restlessness, delirium, hæmatemesis, and rapidly diminishing hepatic dulness, whilst the splenic dulness is increased. But there is no fever. Then supervene tremors, carphology, stupor, coma, and convulsions. The pulse is quick and feeble; hæmorrhages are common; the tongue is dry and brown; the bowels constipated; the urine, although not altered in quantity, yields no urea or uric acid, and contains leucin and tyrosin. Death usually supervenes within a week from the first pronounced symptom.

Treatment.—Purgatives, such as sulphate of magnesia or of potash. Promote excretion of urea by diaphoretics or

diuretics. Treat hæmorrhages and vomiting on general grounds. In the 'typhoid' state, stimulants (ammonia, ether, &c.) are indicated.

ACUTE HEPATITIS (HEPATIC ABSCESS)

Definition.—An acute parenchymatous inflammation of the liver, generally occurring in hot climates, often in relation with dysentery, and usually terminating in abscess.

Causation.—*Age.*—Twenty to fifty. *Pre-existing Congestion* of the liver. *Climate.*—West coast of Africa, India, China, and other hot countries where malaria prevails. New residents are especially liable to be attacked. *Habits.*—Indolence; intemperance, both in eating and drinking. *Previous Liver Disorders*, such as congestion of liver (active or passive), foreign bodies (gall stones, hydatids).

It may be secondary to some specific fever (small-pox, yellow fever), to pneumonia, or phosphorus poisoning. It may exist concurrently with dysentery, or it may occur independently of that disease. Although hepatic abscess occurs with dysentery in many cases in India and other tropical climates, it is probably a condition independent of that disease, as it has seldom or never occurred in cases of dysentery which have originated in this country. Malaria, and the various forms of 'jungle fever' and the like, are also most important causal factors.

Pathology.—The course of events is similar to that which occurs in inflammation of any other organ. There is first a hyperæmia, followed by stasis of blood in the smaller capillaries, and exudation of leucocytes into perivascular tissues. The liver cells swell, become cloudy, lose their sharp outline, their nuclei proliferate, and, finally, they become fatty or pigmented. The organ changes its colour, being first red, then turning to yellow or a dirty white tint. The inflammation may (rarely) subside at this stage, and the liver tissue recover; but if the inflammation go on to abscess, there is a circumscribed greater intensity of inflammation. The patch then becomes opaque, soon softens, the connective tissues of the part break down, and, with the migrant leucocytes,

form pus. The abscess thus formed is surrounded by a girdle of red, congested tissue. One or more foci of inflammation may be the starting-point of a large, circumscribed abscess (tropical abscess). Or the various areas of inflammation may remain discrete, and so form multiple diffuse abscesses (probably pyæmic).

Symptoms are rather vague at first. The liver is enlarged, with fulness in the right hypochondrium. There is pain over the organ, increased by pressure, movement, palpation, deep inspiration, or coughing. The nearer the inflammation is to the surface, the more acute is the pain, and it is often reflected to the right scapula or arm. There is slight fever (102°); jaundice is common; cough and hiccough are frequent. The spleen is rarely enlarged. Vomiting occurs, attended by a coated tongue, irregular action of bowels, and other signs of gastro-intestinal disturbance. When suppuration has supervened, the fever is higher, and presents frequent exacerbations and remissions; it is also accompanied by rigors, hectic fever, and sweatings. There is a bulging, fluctuating tumour in the hepatic region, which may extend in various different directions, according to primary site and to anatomical relations. The final stage is marked by a dry, brown tongue, sordes, subsultus, and the typhoid state.

The abscess may point and rupture, either externally, or into the peritoneal cavity, or into the pleura or pericardium—in short, into any hollow organ or cavity with which it is in contact. There is no rule; it bursts where there is least resistance. Again, it may remain quiescent for months, or become encysted and undergo calcareous change.

Treatment.—*Diet* should be non-irritating, and consist of milk, eggs, farinaceous puddings. No alcohol should be given unless urgently required. Apply hot fomentations, leeches, or cuppings over the liver. *Medicinal.*—Give opium to relieve pain; then relieve the portal congestion by saline purgatives, such as sulphate of potash or magnesia, saline purgative waters (Friedrichshall, Püllna). Chloride of ammonium and the mineral acids are also useful. Ipecacuanlia is indicated if there be any history of dysentery. Avoid mercurials.

When Suppuration has occurred.—Diet to be generous.

Maintain the patient's strength by tonics, mineral acids, quinine.

Aspirate as soon as the presence of pus is evident. Use a small needle. The dangers of puncturing the liver in cases of hepatic abscess are probably overrated. Remember the abscess may be deep-seated, and the first plunge of the needle may fail to find the suppurating cavity. If adhesions have formed, evacuate the abscess freely ; then wash it out, and drain antiseptically.

CIRRHOSIS OF THE LIVER (HOBNAILED LIVER, AND GIN-DRINKER'S LIVER)

Definition.—A chronic atrophy of the liver, due, in the great majority of cases, to alcoholic excess ; characterised by uniform increase of the connective tissue, causing a hardening and a granular condition of the organ.

Causation.—*Alcohol* chiefly, especially 'nipping.' *Age.*—Most frequent between thirty-five and sixty ; but cases are recorded in children, with no alcoholic history.¹ *Sex* has no predisposing influence.

Pathology.—In early stages the organ is probably enlarged. This is followed by hypertrophy of capsule and tissue framework, which is a derivative of the capsule of Glisson. The new growth, rich in capillary vessels, is increased in a special degree round groups of lobules, as well as round individual lobules, which become compressed and even obliterated. It eventually permeates the lobules themselves to a greater or less extent, and separates the hepatic cells, and compresses the minute capillaries of the portal vein. This overgrowth of fibroid tissue contracts, the organ shrinks and puckers, and becomes sharp-edged. The left lobe is most affected. Occasionally the granulations may be felt through an attenuated belly-wall.

A hypertrophic form of cirrhosis is described by most authors. So far as we have been able to investigate the subject, hypertrophy would seem to be the early stage of

¹ *Trans. Path. Soc.*, vol. xxxii. p. 134.

cirrhosis prior to shrinking of the organ. It may last for some months or years, and is usually found in those subjects who have indulged in malt liquors. In spirit-drinkers the atrophic stage is sooner reached.

Symptoms.—The first stage is characterised by discomfort or tenderness in the right hypochondrium, together with thirst, nausea, a foul tongue, flatulence, and other signs of disturbed digestion. When contraction of the liver is established, the liver dulness is much diminished, and the symptoms are such as would correspond to mechanical congestion of the portal

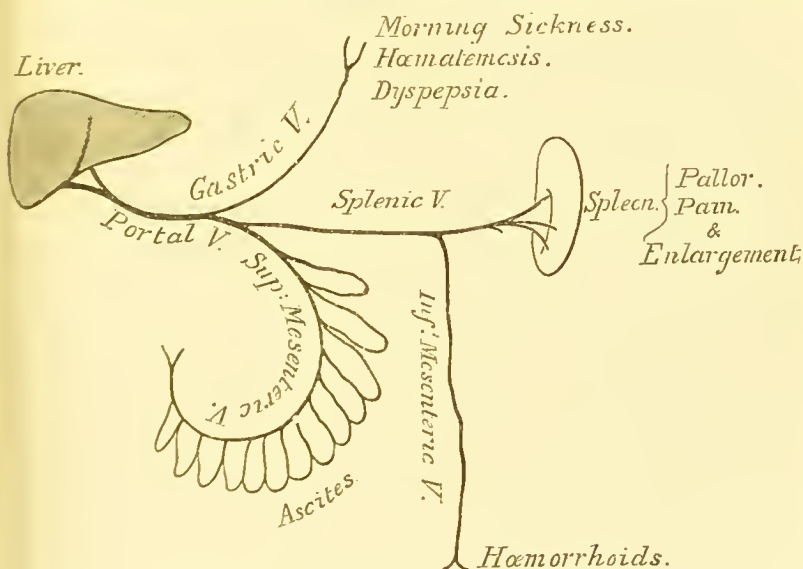


FIG. 10.—TO ILLUSTRATE SYMPTOMS OF CIRRHOSIS OF LIVER

vein and its tributary constituents, viz. the gastric, splenic, superior mesenteric, and inferior mesenteric veins (see fig. 10). Thus we find morning sickness, a distaste for breakfast, hæmatemesis or melena, enlarged and painful spleen, with pallor, ascites or diarrhœa, and hæmorrhoids. There is no regular order for the sequence of the symptoms; but ascites is usually a late sign. As secondary results, there is often engorgement of the general venous system, giving rise to anasarca, distension of veins in the abdominal wall, and venous stigmata in the face. Jaundice is generally an unfavourable

symptom, and occurs towards the end of the disease. The urine is scanty, high coloured, and contains abundant urates ; but the urea is diminished in the later stages, when the secreting structure of the liver is damaged.

Death generally occurs in two to three years after established symptoms are apparent, from exhaustion, or coma (blood poisoning), or hæmorrhage, or from some intercurrent malady, as pneumonia, peritonitis, &c. Still recovery may take place. The gravity of a case is marked by cerebral symptoms, emaciation, ascites, and diminished excretion of urea.

Treatment.—In the early stages diet is most important. Give easily-digested food, such as milk, eggs, soft meats (fish, poultry), farinaceous puddings. An absolute milk and farinaceous diet for twelve months gives good results. Avoid peppers, spices, and rich or oily dishes. Allow no alcohol whatever. The medicinal treatment is to be directed to the relief of portal congestion. Therefore watery purges—*e.g.* sulphates of soda, potash, and magnesia, the aperient mineral waters (Friedrichshall, Hunyadi János), colocynth, podophyllin, compound jalap powder—have been recommended.¹ The saline purgatives are best given while fasting (Murchison). Mercurials, especially the green iodide, are at times useful. Give iodides when the liver is large, with a view to resolution of the recent fibrous growth. Chloride of ammonium is also useful, as it is believed to stimulate the hepatic absorbents. Elaterium (gr. $\frac{1}{12}$), and the dose increased if it can be borne, is also an excellent remedy. Murchison advocated copaiba as giving good results in portal dropsy. When ascites is well established, tonics are said by some physicians to be the only drugs which succeed. Iron, quinine, strychnia, gentian, and the mineral acids may be given.

When ascites is so advanced as to cause embarrassed breathing, albuminuria, or œdema of the legs, abdominal paracentesis must be performed. Do not defer the operation till too late ; indeed, the operation may be done earlier than is generally advocated. The drain of albumin in the large

¹ R. Elaterii gr. $\frac{1}{12}$; Podophyllin gr. $\frac{1}{8}$; Pil : Colocynth : et Hyoscyami gr. iv. Ft. pilula.

quantity of ascitic fluid is no doubt very great ; but, seeing that absorption (the very thing we want) does not occur, the albumin is to all intents practically out of the system, and the sooner it is evacuated, the better the chance of establishing collateral circulation. Besides, pressure on the renal veins and on the cava is removed, and frequently albuminuria and bowel hæmorrhage have been thus arrested.

Probably early and repeated paracentesis gives the best results. (See Cheadle, 'B. M. J.,' vol. ii. 1892.)

FATTY LIVER

Definition.—A condition characterised by the accumulation of fat at the circumference of the liver lobules, causing painless enlargement of the organ. It is generally secondary to some other disease.

Causation.—Indolence with overfeeding, leading to the general deposition of fat throughout the body and organs (liver, heart, kidneys) ; alcoholism ; wasting diseases (cancer, phthisis, phosphorus poisoning, chronic dysentery). In these latter conditions the liver, as it were, becomes the receptacle of the fat absorbed from the other parts of the body.

Pathology.—The liver is uniformly enlarged, soft and doughy in consistence, with a rounded anterior border. It is greasy on section. Its colour is pale yellow and often spotted with patches of light brown tint. The tissue is of low specific gravity, floats in water, and imparts a greasy translucency to paper. Oil is found to accumulate in the circumference of the lobules, attacking the outer zone of cells ; these become enlarged, and lose their shape and characters. The change may be entirely limited to the periphery of the lobules ; but in advanced cases the whole of the lobule may become infiltrated, and form one huge fat cell.

Symptoms.—None very definite. A greasy, smooth skin with a waxy, anæmic tint has been described as a characteristic sign. We find, besides, anæmia and languor, gastrointestinal disturbances, such as dyspepsia, with flatulence and irregular bowels (constipation generally). The liver is

enlarged, without pain. There is no portal obstruction, therefore no enlargement of spleen and no ascites. Jaundice is absent. Syncope, with a weak cardiac impulse, may occur, not so much as part of liver disease, as showing a similar affection of heart muscle.

Treatment.—Diet and regimen are imperative. Avoid fat and fat-forming foods. Give a spare diet, principally of fish and green vegetables. Avoid fermented liquors. Gentle exercise in open air is to be encouraged. Massage, to give tone to the muscular system, is often beneficial. Ensure a regular action of the bowels.

For medicines, give liq. potassæ and the salts of the alkalies, with vegetable tonics. Iron, with strychnia, is useful if there be pronounced anæmia. Ferruginous waters, therefore, are often serviceable. If circumstances allow, a course of Carlsbad or Bath waters is often beneficial.

LARDACEOUS DISEASE OF THE LIVER (WAXY, AMYLOID DEGENERATION)

Definition.—A painless enlargement of the liver, due to infiltration of the walls of the smaller blood-vessels with a transparent, structureless material, as a part of a general condition secondary to prolonged suppuration, or to diseases which cause impairment of nutrition.

Causation.—*Sex.*—Males are oftener affected than females. *Age.*—More frequent in youth than in adolescence. *Prolonged Suppuration*, as in tertiary syphilis, tubercle, empyema, disease of bones and of joints. *Wasting Diseases.*—Cancer, chronic dysentery, ague.

Pathology.—The liver is uniformly enlarged, often three or four times its normal size and weight; it is smooth and pale, with its anterior edge blunt and rounded. On section the cut surface has a pale yellow or tawny tint, with a shiny, glistening appearance. Its consistence is firm, not unlike bacon. It readily responds to the iodine and the sulphate of indigo tests, the lobules with the former reagent changing to a mahogany brown or even a black colour, and a dark blue with the latter.

Microscopically the change is seen to commence in the zone intermediate between the circumferential and the axial vessels ; in other words, in the capillaries of the hepatic artery. Thence it extends, first to the centre, then to the periphery, till the whole lobule is involved and its contents destroyed. The walls of the capillaries become thickened, their histological elements vanish, and eventually the vessels are transformed into tubes having a single coat of waxy or jelly-like material and consistence. The liver cells also take on similar changes. They enlarge, lose their nuclei, coalesce, and become like tears of transparent gum. The lobule may thus maintain at least its original size, or even become greatly enlarged ; but all its component parts are obliterated, their places being occupied by this vitreous material.

Symptoms.—There is a history of a previous condition—*e.g.* suppuration—predisposing to lardaceous disease. There is a non-elastic, painless enlargement of the liver, the hepatic dulness in some cases extending to the iliac crest. The enlargement is slow and gradual. No signs of portal obstruction exist, unless the glands in the transverse fissure be infiltrated ; hence there is no ascites. Marked jaundice is rare, although often an icteroid tinge of conjunctivæ and skin is noted ; but there are general cachexia and anæmia, but no fever. We also look for a similar condition in spleen, kidneys, and intestines, producing respectively splenic enlargement, profound anæmia, and much pale urine of low specific gravity, containing a large amount of albumin and some tube-casts. Occasionally obstinate diarrhœa with offensive evacuations occurs.

Treatment should be mainly directed to the primary cause. Therefore arrest suppuration. In the early stages a cure, or at least an amelioration, may be looked for. Give iodides and mercurials if there be syphilis, and iron to combat anæmia. Potash salts are recommended (Dickinson) ; also tincture of iodine (Murchison), chloride of ammonium (Budd). Beyond these we may give vegetable tonics, mineral acids, and also Woodhall Spa water.

Complications, such as diarrhœa, dropsy, uræmia, must be treated on general grounds.

MALIGNANT DISEASE OF THE LIVER

Definition.—The development, whether primarily or secondarily, of malignant new growth in the liver, producing painful enlargement of that organ, and usually running a rapidly fatal course.

Causation.—*Age.*—Mostly after forty-five, although cases of sarcoma in young children have been recorded by Bristowe and others. There is often a history of those general conditions which predispose to malignant new growth—*e.g.* anxiety, worry, strain, irritation, injuries, &c. *Heredity* is often a strong predisposing factor.

Pathology.—In most cases malignant disease of the liver is secondary to malignant disease elsewhere, especially of structures which are in anatomical relation, or physiologically connected, with the liver. Malignant disease may, however, occur primarily in the liver, especially in people who have been the subjects of gall stones. The new growth is most frequently cancer (scirrhus or encephaloid). Sarcoma is occasionally found in early life. The liver substance may be generally infiltrated with cancerous growth. The organ is then enlarged. But usually it is mapped out into large or small masses with no limiting membrane, but which often coalesce. These masses are depressed in the centre, and vary, as regards their hardness, according as the new growth is scirrhus or encephaloid. When the masses approach the surface, the liver is lobulated and irregular, the irregularity of outline being easily felt through the belly-wall. The centre of the new growth varies also in colour, according to the amount of extravasated blood, medullary cancer being more vascular than scirrhus. Hemorrhage is also easily accounted for by the thinness of the capillary walls and the ease with which they rupture.

Symptoms.—The liver is painfully enlarged, and often of irregular, lobulated outline. The enlargement and the lobulation, however, may vary, according as the organ is generally

infiltrated with new growth or attacked in isolated patches. If a lobulation can be felt to be cup-shaped, the diagnosis of cancer is almost positive. Ascites and jaundice may be, and usually are, present, owing to pressure on the portal vein and to obstruction of bile ducts. There is rarely enlargement of the spleen. In addition, the usual concomitant signs of malignant growth—cachexia and wasting—are present. Look also for malignant growth elsewhere, as the disease in the liver is usually the result of secondary deposit. There are also some important general symptoms. For example: tenderness over the liver, with pain extending to shoulders and right arm, or into the abdomen; wasting, which usually *precedes* the jaundice; signs of gastro-intestinal disturbance, such as nausea, vomiting, flatulence, and diarrhœa or constipation. The urine is of high specific gravity, dark coloured, and loaded with urates.

Prognosis.—The prognosis is invariably bad, life being prolonged for a few months only.

Treatment.—Palliative only. The diet should be easily digested and of well-regulated quantity. Give anodynes, such as opium, chloral, bromides, to relieve pain and procure sleep. Treat the stomach and bowel symptoms on general grounds. Chian turpentine has been recommended, but it is valueless. Avoid mercurials.

HYDATID OF LIVER

The liver is more frequently infested with hydatids than any other organ of the body. The right lobe, and towards its posterior aspect, is a favourite site. The cyst may vary in size from that of a marble to that of a Dutch cheese. (See *Tænia echinococcus*, p. 300.)

Symptoms.—Vary, according to the size of cyst. Often there are no symptoms till a projecting tumour is noticed. If the cyst be large, there are pressure signs, with pain in the hepatic region, vomiting, dyspnœa, and perhaps jaundice. The hydatid thrill may or may not be obtained, but is most frequently elicited in small, tense cysts. Dropsies as the result of pressure are rare.

On aspiration the cyst yields a clear fluid, of low specific gravity (1010 about), containing no albumin, but chloride of sodium and hydatid hooklets. These hooklets are about $\frac{1}{1000}$ of an inch long, and are somewhat triangular in shape.

Progress of Case.—The cyst may shrivel up, owing to death of the parasite, and then undergo calcification. It may rupture, either externally, or internally into the peritoneal cavity, stomach, intestine, pleura, pericardium, lung, or into any tube or cavity ; or it may suppurate, and then behave like an ordinary hepatic abscess, tending to point externally, or to discharge its contents into any canal or sac where there is least resistance.

Treatment.—1. Leave it alone if symptoms be not urgent. 2. Aspirate, if tension be great. It is not necessary to withdraw all the fluid. 3. Electrolysis : by passing two electrolytic needles, two inches apart, into the cyst, a moistened sponge applied at varying points acting as a positive pole ; then pass an electric current for ten or twenty minutes. (See Murchison, ‘Diseases of Liver.’) 4. Promote adhesions of sac to abdominal parietes by moxa ; and subsequently evacuate the cyst, and drain. 5. If suppuration has occurred, make a free incision, and drain as in hepatic abscess.

JAUNDICE

Definition.—A yellow staining of skin, conjunctiva, certain excretions, and most of the tissues by bile pigment. It is a symptom rather than a disease. The discoloration varies in intensity in divers parts. The liver itself is often stained most ; the brain, spinal cord, and humours of the eye escape. The staining is much darker, also, in the aged and wrinkled. It appears in the urine first, but remains longest in the skin. The icterus of conjunctivæ must not be mistaken for subconjunctival fat.

Jaundice is usually associated with a bitter taste in the mouth, disordered digestion, great itching of skin ; xanthopsia and xanthelasma are not uncommon. There is also a tendency to hæmorrhages and anæmia. The pulse is usually slow ; the typhoid state and coma supervene in the last stage.

Jaundice always causes emaciation. This is an important point in diagnosis.

Causation.—The subject of jaundice is divided, so far as its causes are concerned, into two great classes, viz.: (a) OBSTRUCTIVE; (b) NON-OBSTRUCTIVE.

(a) OBSTRUCTIVE JAUNDICE.—The fæces are pale, hard, very offensive, and contain much fat.

1. *Gall Stones and Inspissated Bile.*—Most frequently occur in women over forty years of age. There is probably a history of previous biliary colic, or an attack of colic may supervene. It is said to be prevalent amongst people who consume much fat food. It is often associated with some neurosis, such as neuralgia, migraine, asthma, &c. Search the fæces for calculus. The prognosis as to further attacks may vary as the calculus is faceted (showing that it is one of many) or rounded (solitary). There is often a *sudden* disappearance of jaundice when the calculus escapes.

Treatment.—Apply hot fomentations, and give chloral, chloroform, or morphia subcutaneously during paroxysms of colic. Leeches may also be applied over the gall bladder. Belladonna is said to facilitate the passage of a calculus. Saline purges (sulphates of soda, potash, and magnesia) possibly possess solvent powers on the calculus. Very large doses of olive oil have been recently recommended, but in our experience no relief is thereby afforded. Diet should be light and easily digested. The patient, also, should observe abstinence from fatty foods. (See Biliary Colic.)

2. *New Growth.*—It may be either malignant or benign, and may occur either in the liver itself, or in the stomach, bowel, pancreas, or kidney; or it may be represented by secondary infiltration of glands in the transverse fissure, with cancer, syphilis, lymphadenoma, or tubercle, and so cause pressure on the bile duct. Malignant infiltration of glands in the transverse fissure is perhaps most common.

The following points of diagnosis are important. In cancer the age of the patient is usually over fifty years. Emaciation always precedes jaundice. The detection of new growth in stomach, pylorus, liver, or elsewhere, suggests cancer

as the cause of jaundice. In these cases the jaundice never disappears after it has once been established.

The diagnosis of syphilis or lymphadenoma must be made on general symptoms and the history of the case.

Tumours of the pancreas, kidney, or ovary, and abdominal aneurysm, are all rare causes of obstructive jaundice.

Treatment.—In malignant disease treatment can be palliative only. Give anodynes to relieve pain. If syphilis be suspected, give iodides. If the obstruction be caused by a tumour which is removable, the question of surgical interference must be considered.

3. *Catarrh of Stomach or Duodenum.*—It is common in young subjects as a result of injudicious diet ; in adults, from alcohol, or consequent on syphilis, gout, pyæmia, or phosphorus poisoning.

The **Symptoms** are those of gastro-intestinal disturbance, such as furred tongue, loss of appetite, vomiting, epigastric distress, and constipation, but occasionally diarrhœa. The jaundice disappears slowly.

Treatment.—The diet should consist of light, farinaceous foods, with milk ; avoid all fatty foods. Fomentations or sinapisms may be placed over the epigastrium. Saline purges are necessary. Chloride of ammonium is often most efficacious. Iodide of potassium will relieve if gout be the cause. Mercury and iodides are indicated in syphilitic cases. We can assist digestion by giving ox bile as a digestive. Diarrhœa, occurring from putrefaction of contents of bowels, is best treated by creasote.

(b) **NON-OBSTRUCTIVE JAUNDICE.**—The fæces are normal in colour, and contain bile. Leucin and tyrosin are often found in the urine. After a time the bile becomes inspissated gradually, and may cease to flow. The fæces would then resemble those of obstructive jaundice.

1. *Fevers.*—It may occur as a complication in many fevers, notably pneumonia, typhus, remittent, and typhoid. It nearly always accompanies yellow fever. Whatever fever it attends, the ‘typhoid state’ is common, this condition being due to ‘impaired or deranged metamorphosis in blood and tissues, and

retention of those products of metamorphosis which ought to be eliminated by the kidneys.' (Murchison.)

2. *Poisons, Animal*.—Cases are recorded of jaundice supervening after the bite of a snake or of a rabid animal.

Mineral.—Phosphorus, mercury, copper, and antimony are the principal mineral poisons. Numerous cases of jaundice are recorded as occurring amongst workers in these. Virchow thinks that the jaundice in such cases is due to thickened mucous membrane of the duodenum, which is catarrhal, and therefore obstructive to the bile duct.

Diagnosis is to be made on general symptoms and history of exposure to poison, &c. (See Mercurial and Phosphorus Poisoning.)

Chloroform and ether inhalations have also caused jaundice. Cases are recorded by Frerichs, but they are rare.

3. *Acute Yellow Atrophy of Liver*.—This condition is a sequel of parturition, or of typhus or other specific fever, or of phosphorus poisoning. It may occur in localised epidemics. The jaundice is intense; the 'typhoid state' soon supervenes, then ensue delirium, subsultus, convulsions, and coma. Recovery is very rare.

4. *Neuroses*.—Joy, fear, grief, passion, and anxiety may at times give rise to jaundice, by producing some derangement of natural metamorphosis of blood. It is possibly in its causation akin to the disturbance in lactation (quantity and quality) from nervous shock. In these cases the jaundice comes on suddenly.

Treatment.—In all cases of non-obstructive jaundice diet is important. Give farinaceous puddings and milk. Enjoin regulated exercise, if the condition of the patient permit it. Purgatives are usually required, such as salines, mercury, podophyllin.

But in most cases the jaundice is of secondary importance to the disease or condition which it accompanies. Being symptomatic of a disease or condition, we treat that disease or condition. In acute atrophy it is necessary to maintain the patient's strength by diffusible stimulants, such as ammonia, alcohol; to promote the elimination of effete products by the

skin, bowels, and kidneys; and to treat the 'typhoid state,' which soon supervenes.

TABULAR VIEW OF CAUSES OF JAUNDICE

A. Obstructive (fæces clay coloured)	1. Gall stones and inspissated bile { in common duct in radicles of ducts			
	2. New growth	Malignant	of liver itself secondary infiltration of glands in transverse fissure of stomach of pylorus of duodenum of pancreas of kidney	
			Non- malignant { Syphilis Lymphadenoma	
	3. Catarrh of stomach and duodenum			
	4. Abdominal aneurysm			
	5. Hydatid cysts			
	6. Accumulation of fæces			
	7. Ovarian or uterine tumours			
8. Perihepatitis				
B. Non-ob- structive (fæces nor- mal in colour)	Fevers	{	Yellow fever	
			Typhus	
			Scarlet "	
			Relapsing,,	
	Poisons	Animal	{	Snake bite
				Pyæmia
		Chemical	{	Phosphorus, Copper
				Mercury, Antimony Chloroform, Ether
Acute atrophy of liver				
Neuroses—Joy, grief, fear, passion				
Cirrhosis of liver in its later stages				

OBSTRUCTION OF BILE DUCTS

This condition may occur in any part of the bile passages, whether it be in common duct, cystic duct, or hepatic duct and its tributaries.

Causation.—(a) *Obstruction from without*, such as new growths (syphilitic, malignant, &c.) in the liver, especially its transverse fissure, in the small omentum, stomach, pancreas, or other neighbouring organ. It may also be due to aneurysm of adjacent blood-vessels, to thickening of Glisson's capsule, or to perihepatitis.

(b) *Obstruction from within*.—Gall-stones, parasites (hydatids, distoma hepaticum), chronic catarrh, and inflammatory thickening of mucous membranes of ducts, cicatrix of duct in its course or at its opening into duodenum.

Pathology.—The pathological changes may be subdivided into : (a) changes occurring in the bile ducts and in the lobules ; (b) changes in the characters of the secretion.

(a) As regards the bile ducts, they become dilated above the seat of obstruction. The distribution of the changes in the ducts will then vary in extent according to the site of the obstruction, being universal if it occur in the common bile duct, or local if it occur only in one of the minor subdivisions of the duct. Thus the calibre of the bile ducts may vary in size, sometimes enlarging sufficiently to admit a finger. These channels may eventually undergo inflammatory thickening, or ulceration with the formation of abscess, having ramifying branches following the course of the dilated ducts ; or the ducts may shrivel and their walls become thin, and rupture, discharging their contents into the peritoneal cavity.

The liver tissue becomes deeply olive-green stained ; the whole gland itself enlarges and becomes firmer ; and, according to some authors, there is thickening from inflammatory growth, which, starting from the obstructed bile ducts, pervades the whole liver and causes a hard consistency and roughened surface of the gland akin to cirrhosis. Subsequently the gland shrivels and wastes. The liver cells become stained with bile pigment ; finally, fatty changes and complete atrophy supervene, as in acute yellow atrophy.

(b) The bile alters in colour and consistency. It is generally thin and green-stained ; but it may become thick, and vary in tint as it is mixed with blood, pus, or biliary pigments. In the later stages the secretion may cease to be formed at all.

Symptoms.—Jaundice is the primary symptom, and beyond this there are generally the signs or history of the disease or condition which has given rise to the obstruction. (See Jaundice)

The gall bladder is often enormously enlarged if the obstruction be in the common bile duct, and it can then, in thin subjects, be easily felt through the abdominal parietes. The liver is enlarged and painful, but ascites and splenic enlargement are uncommon. The other symptoms will vary according to the cause of the obstruction. Thus, if jaundice be preceded by biliary colic, gall-stone is indicated ; if preceded by emaciation in a person of advanced life, malignant new growth must be suspected. On the other hand, a history of gastro-intestinal catarrh would point to thickened mucous membrane of the gall ducts. There is no fever. But the concomitant signs of obstructive jaundice are usually present at one time or another. There is high-coloured urine ; clayey, offensive stools ; itching of skin ; xanthelasma, petechiæ. Death supervenes sooner or later, unless the obstruction be removed. Emaciation, bile poisoning, hæmorrhage, as direct sequels, or pneumonia or some other secondary malady are the most frequent causes of death.

Treatment must be directed towards the primary cause. Then on general grounds attend to the state of the stomach and bowels. The diet should be light, nutritious, and easily digested. Check diarrhœa or constipation, and in the latter condition give mild aperients only. The elimination of bile as an excrementitious product should be directed through the channels of the skin and the kidneys. Baths, diaphoretics, together with warm clothing and gentle exercise, are all of good service. Ox bile has been recommended as a substitute for the natural, retained secretion. The question of operation for removal of the cause of obstruction is a surgical one ; but the elements of success depend on the site, nature, and duration of the obstruction.

BILIARY COLIC

Definition.—Attacks of pain of an agonising character, occurring mostly in paroxysms, and due to the passage of gall-stones through the hepatic, cystic and common bile ducts.

Causation.—The method and stages of formation of a gall-

stone are still obscure. There is generally a nucleus, however, which may consist of bile pigment, or some foreign body, parasite, epithelium, or old blood clot. A sedentary mode of life, and any obstruction to the flow of bile, are thought to predispose to gall-stones. Women are affected oftener than men. It occurs more frequently late in life. Obesity is a frequent concomitant condition. The disease is prevalent in Central Europe, where the inhabitants consume much fat (suet) food. Diabetic patients also suffer from gall-stone, as many as fifty per cent. being affected.

Pathology.—Biliary calculi vary in size from coarse dust to that of a pigeon's egg, or even larger. If multiple, they are faceted from mutual attrition ; if single, it is rounded, elliptical, or branched, and closely fits the gall bladder or the duct which contains it. They vary in tint from a tawny yellow to a dark red and even deep black. They frequently float in water, especially if dried ; but oftener their specific gravity just exceeds that of distilled water. Chemically a calculus consists mainly of cholesterin, bile pigment, bile salts, and a little phosphate or carbonate of lime or soda ; but in different calculi the composition varies. Some are formed entirely of bile pigment ; others of cholesterin held together by a kind of cement ; whilst a third variety may contain nothing but pure cholesterin. Their nuclei consist generally of bile pigment, the bile itself being less alkaline than usual.

Frequently gall-stones give rise to no symptoms, and are only discovered after death. But they may cause inflammation and suppuration of the biliary canals, terminating in ulceration and escape of the calculus into the intestinal tube, or into the peritoneum, with fatal peritonitis. It is also probable that, by the irritation which they set up, they may be the starting-point of malignant disease of the liver. If inflammation does not occur, distension of the biliary canals behind the calculus always supervenes sooner or later. Thus, if it be arrested in the cystic duct, the gall bladder may be distended to an enormous size ; if the stoppage occur in the hepatic duct, the biliary canals in the liver are dilated ; whilst it is obvious that, should arrest of the concretion occur in the common duct,

both the hepatic ducts and the gall bladder will equally share in the distension.

Symptoms.—An attack of biliary colic generally commences suddenly and shortly after a meal. The pain is intense, agonising, usually referred to the epigastrium, but at times directly to the site of the gall bladder; it is also paroxysmal, but may still continue, though much diminished, in the intervals between the attacks. The attack is also accompanied by shivering, nausea, and often vomiting. There is an absence of bile in the evacuations, if the obstruction be complete; this causes consequent dyspepsia, jaundice, and constipation. Faintness and even fatal syncope may occur. Occasionally the attack is attended by some slight feverishness.

The entire cessation of pain probably indicates the escape of the stone into the duodenum, and then it will be ultimately found in the fæces. On the other hand, it may return to its first lurking place and cause a repetition of symptoms. If the stone be a large one, and makes its way into the intestine, subsequent smaller ones may pass with little or no signs.

Jaundice, as above stated, is nearly always present at one time or another; but it should be remembered that obstruction must have been of some duration before it does occur.

Treatment.—Prophylactic. The patient should lead a healthy life, and avoid fat foods and over-feeding. He should take gentle exercise. Fermented liquors are said to be injurious. Potash and soda salts, also Castile soap, are advocated with a view to their solvent effects. Phosphate of soda (5j. doses) is the most efficacious. Heroic doses of olive oil have recently been prescribed, but with doubtful effect. (See Goodhart, 'B. M. J.' Jan. 1892.)

During an attack of colic give anodynes. Morphia (by the mouth or by hypodermic injection), belladonna, chloroform inhalations, one or other should be given. Hot fomentations over the liver and epigastrium, poultices, sinapisms, blisters, also assist in relief.

In some cases we may have to advise cholecystotomy, or other surgical operation for the removal of gall-stone.

DISEASES OF THE PANCREAS

ANATOMY

The **Pancreas** weighs about three ounces. It is situated in the abdomen, behind the stomach, and lies in the right lumbar, the epigastric, and left hypochondriac regions.

It rests on the vertebral column, the crura of the diaphragm, the abdominal aorta and inferior cava, the splenic vein and the commencement of the vena portæ. The cœliac axis projects from its upper border, along which also runs, horizontally, the splenic artery. Its lower border is in relation with the third part of the duodenum; the superior mesenteric artery and vein, and the inferior mesenteric vein pass between the pancreas and the duodenum.

The head is embraced by the three parts of the duodenum, with the pancreatico-duodenal arteries and the common bile duct intervening.

The tail or left extremity extends to the spleen, and lies on the left kidney.

The gland is behind the posterior layer of the transverse meso-colon.

Congestion.—The pancreas is subject, as other abdominal organs, to Congestion, as a sequel of chronic diseases of the heart and lungs.

Inflammation of the pancreas has been described as occurring in typhoid fever, pyæmia, and other febrile states. In these conditions the inflammation often advances to a certain degree only and then subsides; at least, such would seem to be the opinion of most authorities—an

opinion which is apparently based on the observations of many cases of fever in which, in a certain proportion of cases, the inflammation does not extend beyond the stage characterised by cellular proliferation and infiltration of the glandular connective tissue with leucocytes, and cloudy swelling of the secreting cells. In some other cases the inflammatory process goes on to the formation of pus and abscess.

A *chronic* form of inflammation of the pancreas has been observed. It is usually an affection secondary to inflammation of peritoneum or surrounding structures, to alcoholic excess, to syphilis, and to diabetes. The organ is hard and shrunken. Its loose connective tissue is increased and thickened, and the interlobular septa also proliferate. The condition is comparable to similar interstitial changes seen in chronic Bright's disease.

Calculus.—The chief duct of the pancreas may be the seat of calculi and concretions mostly composed of carbonate of calcium. As a result of the obstruction which they cause, the duct behind and its tributaries become dilated by accumulated pancreatic fluid, and eventually one or more cysts may form, differing much in size, and, therefore, varying in the severity of the pressure symptoms which they cause.

Hæmorrhages may occur into the body of the pancreas, either from active hyperæmia of its vessels, or from injury, or from passive mechanical congestion as in valvular disease of the heart.

Cancer of the pancreas has been recorded from time to time. The scirrhus form is most frequent. It is generally a secondary deposit; the primary growth being situated at the pylorus, in the stomach, or in the liver. Primary malignant growth in the pancreas itself is sufficiently rare to be a pathological curiosity.

The organ is also subject to **Lardaceous** and to **Fatty Degenerative Changes**.

Symptoms.—The symptoms of disease of the pancreas are obscure and ill-defined. It is plain, from the anatomical relations of this organ, that any increase in its bulk, or any marked alteration in its structure and its vascular arrangements, must lead to pain and possibly to the formation of an

obvious tumour. The pain is described as deeply situated in the epigastrium, of a gnawing or burning character, and extending through to the back and interscapular region. The tumour also would be situated in the epigastrium, possibly in the middle line, but more probably to the right or to the left. It might cause ascites by its pressure on the portal vein. There would also be thirst, loss of appetite, and other signs of slight feverishness. In those conditions of the pancreas which are accompanied by diminution or arrest of its secretion, the stools are described as being pale in colour and infiltrated with undigested fat.

So far as our observation goes, however, there are no diagnostic signs of pancreatic disease. It has no leading symptom to guide us, like bronzing of the skin in disease of the suprarenal bodies ; and what symptoms are present, with the exception perhaps of fatty motions, may very easily be attributed to disease of neighbouring organs.

Treatment.—Very little that is satisfactory can be said on this subject. Inflammation and congestion of the organ must be treated on general grounds as in similar disorders of other glands. The same may be said of lardaceous and fatty degeneration.

Cysts, tumours, and calculi are at times capable of being removed by surgical operations, such as puncture or excision, after opening the belly. Indeed, it is often only after a laparotomy that a correct diagnosis of these conditions has been made.

INTESTINAL WORMS

There are at least a dozen different varieties of worms which are parasitic in the human intestinal canal. In the succeeding pages we propose to speak of the commoner kinds only.

Causation.—The ova may be introduced by water, by food, and perhaps inhaled into the mouth, or carried there by the fingers, or deposited on the lips by dogs or other pets which are permitted to lick their owner's face.

Amongst predisposing causes may be mentioned feeble health, derangements and debility of the digestive functions, the strumous diathesis. In certain warm climates, parasitic invasions are endemic ; whilst hydatid disease, not strictly an intestinal parasite in man, is extremely prevalent in Iceland, the Australian colonies, and other parts.

Intestinal worms are classified according to their shape and form into—

A. TAPE WORMS.—Tæniada or Cestoda.

B. ROUND WORMS.—Nematoda.

A. Tapeworms (TÆNIADA)

All present certain features in common. Thus they are flattened ; they present a head or *scolex* which is armed with suckers ; they have segments (proglottides) which are distinct, separable, sexually complete, and produced by a series of buddings from the head ; they all pass through two stages of existence—viz. (i) the cystic or bladder-worm stage (*cysticercus* or immature worm), during which it infests the tissues or solid

organs of its host ; and (ii) the mature or tapeworm stage, when it is found in the intestinal tube of some other creature. Hence there is a primary host or supporter, generally a flesh-eating animal ; and an intermediary host, generally a vegetarian. To recapitulate. The mature or fully developed parasite possesses—

1. *Scolex* or head, armed with suckers, or hooklets, or both.

2. *Neck*, which is elongated, and finely wrinkled.

3. *Proglottides* or joints, each containing complete male and female generative organs, with a genital pore opening alternately at opposite sides.

STROBILUS is the complete worm ; or it is rather a colony of worms than an individual.

The immature worm presents—

A *cyst* or *vesicle*, containing clear fluid.

A *cystic scolex* or head.

A *neck* finely wrinkled, as in the mature form.

At least three different varieties of mature tapeworm infest the human intestinal canal (generally the small intestine)—viz. *TÆNIA SOLIUM*, *T. BOTHRIOCEPHALUS LATUS*, *T. MADIOCANELLATA* ; whilst one immature worm, at least, the cystic variety of *T. echinococcus*, may exist in our tissues.

TÆNIA SOLIUM

Characters.—Length six to twelve feet : head about one-fortieth of an inch across, somewhat square-shaped with four suckers, one at each corner ; in the centre of the head a beak (rostellum), surrounded by a double crown of hooklets ; neck one inch long, thread-like and wrinkled ; then distinct joints, each containing male and female organs, and possessing a genital outlet behind the transverse middle line. The uterus consists of a well-marked longitudinal canal, with coarse transverse shorter canals opening into it at right angles. As the proglottides become ripe they are shed with the evacuations, and finally rupture ; then they are found to contain ova, each of which has an embryo in its interior. The embryo is armed with a boring apparatus and three pairs of hooklets ; this

being devoured (by a pig generally), enters the host's stomach, then migrates to the solid tissues, and there develops into the cystic worm ('measly pork'). The flesh of this animal is in turn consumed by man, when the parasite loses its vesicular envelope, and attaching itself to the mucous membrane of the intestinal tube develops into the mature tapeworm, and so the cycle of events is again completed.

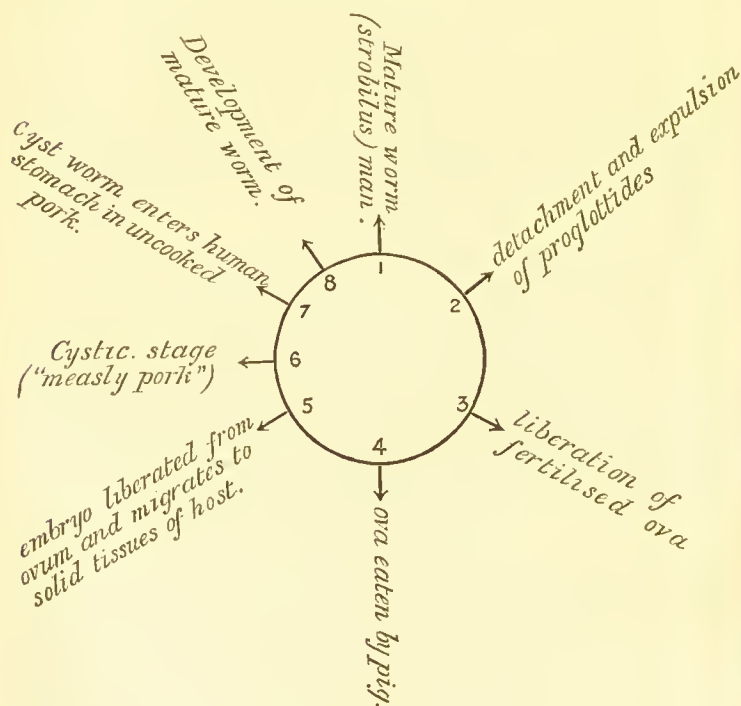


FIG. 11.—DIAGRAM OF CYCLE OF *TÆNIA SOLIUM*

Possibly in some cases man may harbour both the cystic and the mature worm.

TENIA BOTHRIOCEPHALUS LATUS

Characters.—The largest tapeworm infesting the human system, reaching at times thirty feet in length. It has no hooklets. Head oval, with two longitudinal slit-like suckers,

one on each side. Proglottides broader than long. Genital pore in the middle of flat surface of each proglottis ; uterus resembles a rosette. Ova discharged inside the human bowel. Embryo ciliated and aquatic ; intermediary host some fish or mollusc. Somewhat rare in this country, but common in Russia and Holland.

TÆNIA MEDIOCANELLATA

Characters.—A large quadrilateral head, with four large and strong suckers, one at each corner ; absence of hooklets ; the uterus resembles that of *T. solium*, but its lateral canals are finer, and it has in addition a third median canal. The cysticercus infests the flesh of oxen.

Symptoms.—There are no definite symptoms, and the only real evidence of the existence of tapeworm is the discovery of ova or of proglottides in the fæces or on the clothes of the patient. The presence of the parasite may be, however, suspected from the following signs, viz. itching of the nose, pruritus ani, colicky pains and uneasiness in the abdomen, depraved appetite, prolonged anæmia, giddiness, palpitation. Certain neuroses, such as epilepsy, hypochondriasis, have also been observed.

Treatment.—*a.* PROPHYLACTIC.—All flesh foods should be thoroughly cooked, and drinking-water effectually filtered or boiled.

b. CURATIVE.—The administration of indirect anthelmintics or vermifuges is not sufficient to effectually cure the disease ; direct vermicides are requisite. The former class of drugs expel portions of the parasite only ; the latter dislodge the head of the parasite, and thus kill it. The most reliable drug appears to be the liquid extract of male fern given whilst fasting. Prepare the patient as follows : the last meal of the day should be given at about five P.M. ; at bedtime order to be taken an ounce of castor oil to empty the bowels ; at about seven o'clock the next morning give the liquid extract of male fern¹ ; this should be followed about

¹ ℞. Ext : Filicis Liq : ℥xxx. ; Liq : Potassæ ℥x. ; Mucil : Acaciæ ʒj. ; Aquæ Menth : Pip : ad ʒj. Misc.

three hours afterwards by another dose of castor oil, which will expel the dislodged parasite. If after careful search the head of the worm is not found, resume the treatment after an interval of two days. But even this drug is not always successful. Other remedies which may be tried are oil of turpentine,¹ eusso, kamala (gr. xxx.—5j.), alcohol in concentrated and repeated doses. Powdered glass has also been advocated.

TÆNIA ECHINOCOCCUS

Characters.—The mature worm infests the intestine of dogs, wolves, foxes. It is an exceedingly small parasite, seldom measuring more than a quarter to half an inch in length. The head is armed with a double row of recurved hooklets, and four suckers; the fourth segment or proglottis only is sexual. The cysticercus may infest any tissue in man; its favourite habitat is the liver, or subperitoneal tissue. A person harbouring the cystic form of *T. echinococcus* may not inaptly be regarded as a 'measly' man. The cyst may be of indefinite size—from a grape to that of a football. Its walls consist of a delicately laminated tissue, lined internally with a fine cellular or epithelial coat. The surrounding tissue in which the cyst is embedded presents a zone of inflammatory growth. The contents of the cyst are a clear fluid of specific gravity about 1007, containing shed hooklets, chloride of sodium, but no albumin. A characteristic of this larval stage is the formation of daughter-cysts, which may bud and protrude internally to, or externally from, the parent vesicle.

The life of this immature worm may be prolonged to any length of time, or, from some cause or other, it may die, shrivel up, and undergo fatty or calcareous degeneration; it may suppurate, or rupture and cause the death of its host.

Treatment.—**PROPHYLACTIC.**—It is obvious that dogs known to be suffering from the intestinal form of this parasite should be excluded from the house, especially where there are children; nor should dogs be permitted to have access to fields and gardens from which vegetable food is obtained.

¹ R. Ol: Terebinth: ℥xx.; Mist: Amygdale ad 5j. Misc.

Watercresses, lettuces, celery, and other vegetables which may harbour the ova contained in a dog's excrement should be thoroughly washed or cooked.

CURATIVE.—The treatment of the cyst is mainly surgical (see Hydatid of Liver). Unless the presence of the tumour give rise to distressing or dangerous symptoms, it were best left alone. On the other hand, if the cyst be superficial or there be signs of suppuration it should be evacuated.

B. Round Worms (NEMATODA)

General Characters.—They are all elongated and cylindrical, tapering to a point at each extremity. They possess a mouth and an alimentary canal terminating in an anus. The sexes are separate, the females being larger than the males. Some of them, if not all, pass through a larval stage embedded in the tissues of an intermediary host. Some six or seven varieties of nematode worms are known to infest man, the chief being *ASCARIS LUMBRICOIDES*, *OXYURIS VERMICULARIS*, *TRICHINA SPIRALIS*, *FILARIA SANGUINIS HOMINIS*.

ASCARIS LUMBRICOIDES

Characters.—This worm is from ten to twelve inches long, and of a pink or buff colour. The female is much larger than the male, and sheds its ova in large numbers into the small intestine which it infests. The ova are elliptical bodies about $\frac{1}{340}$ of an inch long, having a tough nodular envelope. The worm is migratory in its habits, and may wander to the stomach, into the mouth, through the nares, or into the gall duct of its host. We have seen, passed by a child *per anum*, a boot-button with an ascaris threaded through the looped shank. The parasite is common all over the world, and so far as our experience goes it is much more frequent where dirt, squalor, and surface drainage prevail. For example, we have found almost every third child in an insanitary colliery village suffering from the worm and its effects. Again, in some rows of low tenements we have seen the disease amount almost to periodic epidemics.

Symptoms.—No definite symptoms. Occasionally there

are vomiting and other signs of dyspepsia ; with itching at the anus, especially at night. Diarrhœa and irregularity of the bowels are not uncommon, whilst in children there may be marked fever, with convulsions. Microscopic examination of the *feces* would detect the ova in large numbers.

Treatment.—**PROPHYLACTIC.**—All food should be well cooked, drinking-water should be boiled or effectually filtered ; and possible sources of re-infection from the clothes or the body of the patient should be met by strict cleanliness.

CURATIVE.—Various purgatives will expel the worm, such as aloes, calomel, scammony. Other drugs act as direct vermicides, viz. turpentine, the various preparations of iron, and notably *santonin*.¹ This latter is the best remedy, given in two-grain doses, followed by a mild purge.

OXYURIS VERMICULARIS

Characters.—A minute worm, one quarter to half an inch in length, infesting the *cæcum* and large intestine, especially the lower two-thirds of the rectum. It is migratory in its habits, and consequently may pass through the anus into the vulva, urethra, on to the skin of thighs or buttocks, or the body linen of the patient. The ova, each of which contains an embryo, may thus easily be transferred to the patient's mouth, especially by children who have the habit of sucking their fingers.

Symptoms.—Itching of the anus, and grinding of the teeth, especially at night, itching of the nose, capricious appetite. There are, however, no diagnostic symptoms. The child is generally brought for advice after having passed quantities of the worm in its stools.

Treatment.—Drugs given by the mouth are neither necessary nor effectual. The parasites are easily destroyed and expelled by enemata (repeated if necessary) of infusion of quassia, green tea, salt and water, or weak solutions of tincture of iron. Bodily cleanliness is important.

¹ R. *Santonini* gr. ij.; *Pulv*: *Sacchar*: *Alb*: gr. ij. *Misce*; *ft. pulv.*

TRICHINA SPIRALIS

Characters.—They have two stages of existence : (1) a non-generative form in the muscles of some animal (generally a pig), the worm being spirally coiled in a cyst. The cystic form being devoured by man, the parasite is liberated by its capsule being dissolved ; it then assumes (2) its mature form, develops generative organs, produces ova, each of which throws off an embryo ; these migrate to the various muscles of the body, perforate the sarcolemma, and then feed on the muscle fibre and its juices. The irritation thus set up seems to lead to the formation of a spindle-cell cyst or shroud containing the worm. Finally the cyst wall becomes infiltrated with calcareous matter, and thus destroys the life of the contained animal.¹ Invasion of the human body by this parasite is known as Trichinosis.

Symptoms.—The course of the disease (TRICHINOSIS) is slow and protracted, the symptoms somewhat resembling those of acute rheumatism together with enteric fever. Thus there is fever of a typhoid type, the temperature intermitting, and often rising as high as 104° F. There is pain in the voluntary muscles (not especially in the joints), with stiffness, muscular debility, nausea, and vomiting. If the œsophagus be involved, dysphagia is also present. As the disease progresses it is characterised by emaciation and œdema of the subcutaneous areolar tissue, notably of the face. Death occurs from gradual exhaustion, or from pneumonia or peritonitis. Cases of recovery, however, have been recorded after a lingering illness, varying in duration from one to four months.

Treatment.—PROPHYLACTIC.—Pork suspected of harbouring the cystic form should be rigidly rejected, or at least well cooked. Smoking or other forms of ‘curing’ are not sufficient.

CURATIVE. No known drugs have any curative effect. Our aim should be directed towards maintaining the patient’s strength by nourishing food and stimulants. Give opium or

¹ A male body dissected in the anatomical rooms of St. Thomas’s Hospital was found to be the subject of trichinosis, every muscle in the body, except the heart, being freely invaded by the worm.

other anodyne to relieve the pains. Treat vomiting and other symptoms on general grounds.

FILARIA SANGUINIS HOMINIS (*Lewis*)

Characters.—A nematode worm. Its embryo is $\frac{1}{3300}$ of an inch broad by $\frac{1}{75}$ th long, and hence readily passes along the blood capillaries. The sexes are distinct, the mature male worm measuring about 3 inches long by $\frac{1}{30}$ in diameter, the females being about twice the size. It is common in China and the East; both man and the lower animals are liable to be infested with the parasite.

The embryo is abstracted from the blood of man by a female mosquito or some other blood-sucking insect, and enters into the stomach of its new host, where it becomes furnished with an alimentary canal and a boring instrument. The embryo is then probably deposited in water and is thence conveyed to the human stomach either by the medium of drinking-water, or by some aquatic animal which is consumed as food. From the human stomach it escapes to the lymph vessels, where it becomes sexually mature and breeds. Its embryos, passing along the lymph vessels and blood capillaries, await their turn to be taken up by the puncturing proboscis of the mosquito.

Symptoms.—Often none whatever. Infection of the lymph vessels appears to be quite compatible with good health. In others the invasion is characterised by fever of an irregular intermittent type, accompanied by inflammation of the lymphatic vessels and induration of the glands. Accompanying these symptoms there may exist elephantiasis of scrotum or legs, and chyluria, either separately or together. The lymph escaping from a punctured vessel contains embryo filariæ. The onset of symptoms appears to be dependent on the obstruction to the lymph current caused by glands which are indurated and inflamed by the accumulation of embryos. As a result of the obstruction, the lymph vessels become engorged and distended with lymph, and produce chyluria or lymph scrotum, or both, according as the site of the obstruction is in the kidneys or groins.

The activity of the embryos in the human blood is confined entirely to the hours of dusk. They apparently disappear or hide during the daytime. They are thus described as nocturnal in their activity, corresponding with the period of attacks of the mosquito.

Treatment.—Removal from infected district. No medicinal remedy is known. Quinine in large doses appears the most rational remedy.

DISEASES OF THE RESPIRATORY SYSTEM

ANATOMY

The TRACHEA is about $4\frac{1}{2}$ inches long in the adult. It commences opposite the sixth cervical vertebra, and extends downwards into the thorax, and divides opposite the third dorsal vertebra into right and left bronchi.

In the NECK it is overlapped by the thyroid gland, the isthmus of which lies on the second and third rings ; by the lobes of the thyroid ; by the inferior thyroid veins ; and by the sterno-mastoid, sterno-hyoid, and sterno-thyroid muscles.

Behind is the œsophagus, with the recurrent laryngeal nerves lying between these two tubes.

Laterally are placed the lobes of the thyroid gland, and the carotid sheaths, each containing the common carotid artery, the internal jugular vein, and the vagus nerve.

In the THORAX in front are the arch of the aorta, with its innominate, left carotid, and left subclavian arteries, the left innominate vein, the bifurcation of the pulmonary artery, and the deep cardiac plexus.

Behind is the œsophagus.

Laterally, on the right, are the innominate artery, the right innominate vein, and the right phrenic nerve.

On the left are the left carotid and subclavian arteries, and the left phrenic nerve.

The BRONCHI, right and left, result from the bifurcation of the trachea. The right bronchus is nearly horizontal in direction ; the left runs more obliquely downwards to emerge under the arch of the aorta. The septal cartilage which

divides right from left bronchus is not in the middle line ; it is more to the left, and thus favours the entrance of any foreign body into the right bronchus in spite of its more horizontal position.

The *Right Bronchus* is one inch long.

The *Left Bronchus* is two inches long.

The *Trachea* bifurcates at the third dorsal vertebra.

The *Right Bronchus* enters the lung at the level of the fourth dorsal vertebra.

The *Left Bronchus* enters the lung at the level of the fifth dorsal vertebra.

The *Right Bronchus* runs behind the right auricle, the superior vena cava, and the right division of the pulmonary artery. The vena azygos major vein arches over it from behind to enter the superior vena cava.

The *Left Bronchus* passes beneath the arch of the aorta and the left pulmonary artery, and lies over the œsophagus, the thoracic duct, and the third part of the aortic arch.

The LUNGS are conical-shaped organs, each weighing about twenty ounces. The right, however, is the larger, although shorter on account of the higher ascent of the diaphragm on the right side.

Each lung presents an outer and an inner surface, a base and an apex, an anterior and a posterior border. From about two inches from the apex—or to be more exact at the level of the third dorsal spine—a fissure runs downwards and forwards, and terminates near the lower end of the anterior border. But it varies considerably on the two sides, and in different bodies.

Each lung is thus unequally divided into two great lobes, an upper and a lower, the latter being much the larger.

The outer surface is convex, marked by impressions of the ribs, and entirely invested by pleura.

The inner surface is concave, for the reception of the heart and pericardium. It presents a hilum, which is situated a little above and behind the middle.

The base is concave, and corresponds accurately with the convex surface of the diaphragm. It is surrounded by a thin edge, which fits between the ribs and the diaphragm.

The apex reaches about an inch and a half above the first rib, under cover of the scalenus anticus, a layer of the deep cervical fascia being prolonged from this muscle on to the summit of the parietal pleura. The apex has also the arch of the subclavian artery a little anterior to it.

The root of a lung consists of pulmonary vein, pulmonary artery, and bronchus, in that order from before backwards; held together, with branches of the sympathetic nerve and lymphatic glands, by the reflection of the pleura. The root of the right lung passes behind the superior cava, the right auricle, and the right phrenic nerve; whilst the vena azygos major vein arches over it from behind to enter the superior cava.

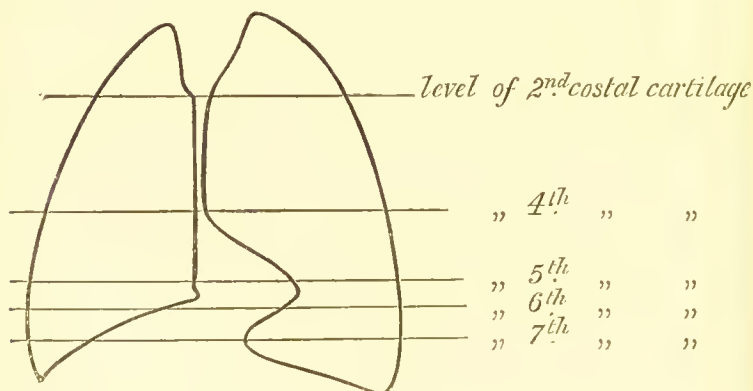


FIG. 12.—DIAGRAM TO SHOW RELATION OF LUNGS TO THE FRONT OF THE CHEST

The root of the left lung lies on the third part of the arch of the aorta, the oesophagus, and the thoracic duct. It is crossed anteriorly by the left phrenic nerve.

Probably the most important structures in the roots of the lungs, from a medical point of view, are one or two lymphatic glands.

The anterior border of each lung is thin, and lies immediately under the sternum. On the right side it extends from the junction of the first with the second pieces of the sternum, down the middle of this bone to the level of the sixth costal cartilage, where it then meets the sharp border of the

base. On the left side this anterior border, commencing at the same place, extends down the middle line of the sternum as far as the level of the fourth costal cartilage only ; thence it runs outwards to the outer end of the fifth costal cartilage ; and again turns inwards to the middle of the seventh costal cartilage, where it joins the circumferential border of the base, as on the right side. It will thus be seen that this border of the lung presents a V-shaped notch, the apex of which is outwards and downwards. In this notch or space part of the heart is seen, principally the right ventricle (see fig. 12).

The *MEDIASTINA* are spaces in the middle line of the thorax, between the sternum and the vertebræ.

The anterior mediastinum is that space between the sternum and the heart and pericardium. It contains the internal mammary artery, some lymphatics, loose areolar tissue and fat, and the *triangularis sterni*, together with the origins of the sterno-hyoid and sterno-thyroid muscles.

The middle mediastinum contains the heart and pericardium, the first part of the aortic arch, the pulmonary artery, the superior vena cava, and the phrenic nerves.

The posterior mediastinum, the space between the heart and the backbone, holds the third part of the arch of the aorta, the trachea, the œsophagus, the thoracic duct, the vagi nerves, the azygos veins, and the sympathetic chains of nerves with their splanchnic and other branches.

The superior mediastinum is formed by the divergence of the summits of the two lungs. It lies above the horizontal plane of the junction of the first and second parts of the sternum.

It contains the trachea, œsophagus, thoracic duct ; the innominate veins and superior vena cava ; the phrenic, the pneumogastric, and the left recurrent laryngeal nerves ; and the thymus and cardiac lymphatic glands. (Thane.)

The chest is divided into certain regions, the boundaries of which are merely approximately defined ; but for all practical

purposes their situations are denoted by the names. Thus, *anteriorly*, in the middle line, we speak of the suprasternal, the upper sternal, and the lower sternal; at each side again there are the supraclavicular, the infraclavicular, the mammary, and the inframammary regions. The *lateral* regions comprise the axillary and the infra-axillary. *Posteriorly* are the suprascapular, the scapular, the infrascapular, and the interscapular areas, the situations of which speak for themselves.

It were always better, however, where the exact localisation of any phenomenon is to be recorded, that measurements be made from some definite bone, such as a vertebra or a bony ridge; or from some fixed landmark, such as the nipple.

The nipple in the male is at the lower border of the junction of the fourth rib with its costal cartilage.

The junction of the manubrium sterni with the gladiolus can always be felt. It is on a level with the middle line of the second costal cartilage.

The scapula extends from the second to the seventh ribs.

Physical Examination of the Lungs is ordinarily conducted by *Inspection, Palpation, Percussion, Auscultation*, and *Mensuration*.

(A) **INSPECTION AND PALPATION**.—The patient's chest should, if possible, be thoroughly bared and exposed to view. Two important factors in diagnosis of disease of the thorax are thus displayed, viz. its shape and movements. As regards shape, we are thus enabled to ascertain whether the thorax be of the narrow and small type (spare chest), or deep and well-developed, both of these varieties being consistent with perfect health. Or we may detect the narrow chest with flattened clavicular regions, and narrow costal angle in a phthisical subject; and, on the other hand, the deep, bulging thorax, with wide costal angle of emphysema. Distortions of thorax, the result of occupation, or of congenital or acquired disease of the bony walls and viscera, will also be revealed.

Abnormal conditions of veins and skin, local bulges, &c., will also at once be detected.

The inspection of the movements of the chest during respiration are equally, if not more, important.

Note if there be symmetrical movements of both sides, or if in one side the expansions in inspiration be limited or lost, and exaggerated in the opposite side, suggesting impediment to the entrance of air to one lung, whilst to the other it enters in excess (compensation). Similarly, movements of the chest walls may be limited in certain areas, due to some local obstruction to the free access of air to that part.

By PALPATION we are enabled to estimate in great measure the freedom of the respiratory movements of the chest as a whole, whether they be in excess or diminished on one side, or whether the conditions be confined to localised areas or regions. Vibrations in the bronchial tubes, the result of inflammatory thickening and exudation (*râles*), are occasionally transmitted to the chest walls, and rough pleuritic friction may often easily be detected in thin subjects (friction thrill). Vibrations produced by the voice (vocal fremitus) and cough are also valuable diagnostic signs ; they are increased where the lung is solid, and diminished in pleuritic effusion or in thickened pleura.

(B) PERCUSSION.—By sharply tapping on the parietes of the chest (or other cavity) certain musical notes are elicited. The variation in character and intensity of such notes, together with the sense of resistance afforded to the percussion finger, affords most valuable evidence of the condition of its contents.

The note which is thus produced is in a slight measure caused by the vibration of the walls of the chest ; but it is more especially due to the vibration, together with the tension and varying lengths, of the columns of air contained therein.

The methods by which percussion may be practised are (i) the *mediate*, and (ii) the *immediate*. The former is most frequently practised, one or more fingers of the right hand being used as a hammer, and a finger of the left hand or a flat disc of ivory or other material used as a pleximeter.

In immediate percussion the chest walls are smartly struck by the tips of the fingers, or by the flat hand, or by a specially constructed rubber hammer.

For all practical purposes the physician's fingers are quite

sufficient aids to examination by percussion. Pleximeters and hammers are, except in rare cases, entirely superfluous. One or two fingers of the left hand are to be firmly and evenly placed on the chest or part to be examined, and then smartly struck by the pulpy end of the right middle finger. An appreciable musical resonant note is thus produced in those areas of the chest where healthy lung comes to the surface. On the other hand, a dull note is produced by percussion over the sites of solid organs, of tumours, or of effused fluids.

One or two precautions are necessary for perfect percussion. The fingers to be struck should be firmly and evenly placed on the chest parallel with the ribs, so as to allow no air to intervene. Percussion should be made from the wrist only, the movement being free and elastic, somewhat like that seen in the hammers of a piano. Force is not necessary for the production of a good note. Corresponding regions of the chest should be percussed and compared, and similar methods and amount of force employed, as it is quite possible to obtain two different notes from corresponding regions, if on the one side only an excess of force be used, or a pleximeter employed. It is well, also, to remember that with some perfectly healthy lungs there is a slightly increased resonance on the right side, as compared with the left.

It is obvious that a healthy chest containing lung tissue distended to a certain degree by the atmosphere will, on percussion, give forth a sonorous note, excepting such parts of the chest against which liver, heart, or other solid bodies impinge, and where the percussion note would be dull. We thus speak of normal resonance and of normal dulness, and variations from these healthy standards are significant of local disease.

Abnormal Percussion Sounds.—Normal resonance may be impaired only—that is to say, the percussion note is not so resonant as it should be, but still not sufficiently flat to constitute dulness. The conditions giving rise to this are numerous. Amongst the most important are (1) imperfect expansion of the chest ; (2) congestion of lung tissue ; (3) slight effusions ; and (4) general deposit of tubercle.

Resonance may be increased in varying degrees of intensity. The hyper-resonance may be *general* in emphysema of the lungs, and especially in cases where there is perforation of the pleura and subsequent collapse of lung from equalisation of the atmospheric pressure (pneumo-thorax) ; or it may be *local* only, from patches of emphysema, a limited pneumo-thorax, or by the action of effused fluid compressing the lung tissue into a limited area, usually the upper and inner regions of the thorax (Skodaic resonance). It may also be caused by an inflated stomach or bowel.

Another abnormal percussion sound—the ‘cracked pot,’ or ‘bruit de pot fêlé’—is heard in cases of cavity (especially at the apices of the lungs) or in dilated tubes. For its perfect production it is necessary that the patient’s mouth be open ; that percussion be sharp, and performed during the act of expiration ; that the cavity be near the surface, but with a layer of crepitant lung tissue overlapping it ; and that the chest wall be elastic and yielding. It is, however, not infrequently heard in percussing the chests of perfectly healthy children.

The sound may be imitated by percussions on an ordinary empty match-box.

Normal dulness, as before stated, occurs in those parts of the chest where heart, liver, and bodies more solid than lung, impinge. But such areas of dulness may be also *diminished* by disease in which (1) the lung tissue unduly overlaps and displaces these solid organs (emphysema) ; (2) or in which it is displaced by air (pneumo thorax). On the other hand, we speak of dulness being *increased* in cases of marked consolidation of lung (tubercular deposit, new growth, pneumonia) ; and *greatly increased* so as to constitute ‘tubby’ dulness in effusions into pleura or pericardium.

Percussion also yields another physical sign of extreme value, viz. the sense of resistance. It is obvious that with a pleura distended with air, or lung tissue that is largely emphysematous, percussion will give a sense of undue elasticity—a drum-like sensation as well as note ; whilst a thorax in which the lung is markedly consolidated, or in which there

is much pleural fluid, with high tension, the dull percussion note will be supplemented by a sense of hard resistance which is very characteristic. These signs are of great importance, in that we often gain much information by combining the senses of hearing and touch.

Further, it is as well to remember that percussion varies even in health, according to different circumstances. Thus, it gives a higher and more resonant note in young people, in males, in athletes, and in the erect position with the chest thrown forward.

We may also deceive ourselves by varying the force of percussion over different regions which we wish to compare; or by comparing the note yielded during inspiration, when it will be slightly higher, with that during expiration, when the pitch will be lower.

(C) AUSCULTATION is a term applied to the method by which we recognise sounds occurring in the lungs, heart, and also the various cavities of the body. It may be practised by placing one's ear directly over the part to be examined (immediate auscultation), or by the aid of a conducting medium, called a stethoscope (mediate auscultation).

Stethoscopes are of various shapes, designs, and materials, and it is perhaps needless to say that each physician has a liking for one or other form of instrument with which he is familiar or to which he is accustomed. It may, however, be observed that sounds produced in the respiratory tract are perhaps best heard with the binaural stethoscope, whilst heart sounds are probably easier detected by the simple single tube.

In auscultation of the lungs we must take note of sounds produced by :

a. Respiration (inspiration and expiration), such as their character, relative duration, or their accompaniment by musical or other abnormal noises. It should be remembered that the expiratory sounds are most important.

b. Voice (whispered and spoken) with all its modifications, known as diminished or increased vocal resonance, pectoriloquy, ægophony, &c.

c. *Cough*, which may be considered as a combination of voice and respiration.

NORMAL AUSCULTATORY SOUNDS OF LUNGS.—*a. Respiratory*.—The vesicular murmur is that which is produced by air passing through healthy bronchial tubes into healthy terminal air sacs. It has been likened to the noise of a gentle breeze, or to the sighing of the wind through a tree in summer. It is best marked towards the bases and sides of lungs, that is to say, furthest from large bronchial tubes. Always distinct in children, in thin persons, and in women. It should be well heard on both sides of the chest, and any deviation in intensity, duration, or rhythm is suspicious of disease. In children the breathing is generally arrested for a few seconds during stethoscopic examination.

Bronchial breathing.—The sound produced by air passing through and causing vibrations in a healthy bronchial tube. It varies in intensity from the loud noise heard over the trachea (tracheal breathing) to the sound heard over the large bronchial tubes at the root of a lung. It diminishes, in health, away from the larger tubes ; or, in other words, if we auscultate in those situations where the vesicular murmur is most predominant and can, as it were, drown the somewhat feeble tones in the smaller tubes. Some of the vibrations are also produced at the rima glottidis, and conveyed to the ear by large bronchial tubes and the stethoscope, the one being, as it were, continuous with the other.

b. Vocal.—In a normal chest the voice, produced at the vocal cords, and modulated or modified by the buccal cavity, comes to the ear of the auscultator as a buzzing sound attended by fremitus. It is known as vocal resonance, and best heard over the larynx, trachea, and larger tubes. It is stronger on the right side than on the left, in front than behind, in men than in women, and it is produced better by high than by low notes. Vocal resonance may, in disease, be lost altogether, or greatly exaggerated ; or present various modifications, such as bronchophony, pectoriloquy, egophony, in consolidation of lung tissue, cavity of lung, or pleural effusion respectively.

c. Cough. Respiratory and Vocal.—Consisting as it does of a long inspiration followed by an explosive expiration, cough may also be modified according to the variations in breathing which are mentioned under the various headings, but in a more marked degree. It has also the advantage that it presents differences which may also be present in the voice sounds. We thus, as it were, obtain a double test. If we wish to apply it as a test for vocal phenomena, we listen over the situation of the larger tubes ; if as a test for respiration, over the bases and sides of the lungs.

ABNORMAL AUSCULTATORY SOUNDS IN LUNGS.—*a. Respiratory.*—Modifications of vesicular murmur. 1. *Too loud* (puerile breathing). A harsh, loud character of breathing, in cases where extra work is thrown on the lung or portion of lung, as in compression of bronchial tubes, or collapse of lung, &c.

2. *Too feeble.*—In emphysema, compression of lung by fluid, pleural adhesions, atrophy of chest muscles (senile breathing).

3. *Irregularity of rhythm.*—Varying in degree from ‘jerky’ to ‘cog-wheeled’ respiration. This may be due to pleural adhesions which are not yet firm ; to obstruction in smaller bronchial tubes ; or it may be due to defective nerve impulse (in nervous subjects) ; or, again, to direct cardiac impulse (hypertrophy or aneurysm).

4. *Modifications of bronchial breathing.*—In intensity, over sites where it is usually heard ; and in situation (tubular breathing), when heard over areas in which it is normally absent (consolidation or compression of lung).

5. *Cavernous breathing.*—A loud, hollow, respiratory murmur, bronchial in character, but exaggerated by the glottic sounds reverberating through a hollow cavity or dilated tube. The cavern must be in communication with a patent tube. If the cavity be of extreme size, the sounds are more intensified still, and are then known as *amphoric respiration*.

6. *Moist sounds* may be produced in tubes (large and small), in vomicae, in alveoli of lungs, and in the pleural cavities. They are produced by the passage of air through fluids of

varying density. The sounds may differ in volume, pitch, and in other general characters, and are called, according to their degrees of fineness, *fine crepitations* ; *subcrepitant râles* ; and *râles*.

Fine crepitation, or *crepitant râle*, is of extreme fineness, heard during, and especially at the end of, inspiration. It is a typical physical sign of pneumonia at about the end of the first stage. It is generally supposed to be produced in the air cells only, and resembles the noise made by twisting one's hair between the finger and thumb. Coughing does not remove it, nor alter its character.

Subcrepitant râle.—A crackle, coarser than the preceding, and generated possibly in air cells and smaller tubes. It is proof of the liquid contents of tubes and air cells, and of the expansion and recoil of the latter. It accompanies inspiration and expiration.

Mucous râle.—The coarsest of the three. It may accompany inspiration only, or expiration only, or both. It is produced by the bursting of air bubbles in fluid contained in medium-sized tubes. The frequency of the bubbles, and therefore the character of the râle, varies according to the strength of the respiratory effort, and also with the quantity and quality of the fluid. It may also be so coarse in character when generated in large primary tubes as to give rise to gurgling.

7. *Metallic tinkling* is the distinctly musical character, or splash, which is occasionally imparted to a moist sound of the coarser sort. It is significant of a large cavity, with smooth and thick walls, containing fluid and air.

8. *Pleuritic friction* is produced by the movements of the two surfaces of pleura which have been roughened by inflammation. It may resemble crepitation ; but in the early stage it distinctly suggests a dry, rough surface, as it is quite superficial, and apparently produced immediately beneath the stethoscope.

9. *Succussion* is the sound which is produced by shaking a patient in whom there is air and fluid in the pleural cavity. It is frequently distinctly musical in character, resembling the

sound produced by shaking a leather bottle containing some fluid. Although not a respiratory phenomenon, it may be conveniently mentioned here.

10. *Dry sounds* are produced by mechanical agencies, somewhat similar to those which generate moist sounds, such as air passing through tubes diminished in calibre, by spasm, by peribronchial pressure, by swollen mucous membrane, or by thickened secretion. The sounds vary in pitch, according as they are generated in the larger pipes (sonorous rhonchus) or in the smaller (sibilus, or sibilant rhonchus). *Rhonchus* is, therefore, a note of low tension, produced with inspiration or expiration, but especially the latter, and is best heard in cases of chronic bronchitis, where the elastic recoil of lung tissue is impaired. It has been likened to a snore, or a dove's cooing. The coarser rhonchi may cause a distinct vibration in the thorax, perceptible to the hand placed on the chest (*rhonchus fremitus*). *Sibilus* is a characteristic whistle, audible at some distance from the patient. It is heard in inspiration and expiration, the latter especially.

b. *Vocal*.—Modifications of vocal resonance. As stated before, the spoken voice is most distinctly heard when the stethoscope is placed over the trachea and bronchi and their larger subdivisions, and it diminishes in strength as we listen further away from these larger and comparatively solid-walled conductors, till at the sides and bases of the lungs it becomes a mere buzzing sound.

Vocal resonance may, however, undergo several modifications in disease: (1) It may be increased (*bronchophony*) in situations where it is normally weak, as when patches of consolidated or condensed lung tissue act as good conductors of the vibrations in the trachea and bronchi. Largely dilated bronchi would produce similar effects, the vocal sounds then, however, being attended by a metallic echo, such as is produced by speaking down a trumpet (*amphoric resonance*). (2) On the other hand, it may be *diminished* or entirely lost, when the lung is surrounded by fluid (serum, pus, hydatid, &c.), or in cases of atelectasis, or of collapse of lung (pneumo-thorax, chronic bronchitis). (3) *Pectoriloquy*, another modification of

vocal resonance, is a term applied to that phenomenon heard when the *articulate* voice of the patient is conducted through the chest and the stethoscope to the auscultator's ear. Although generally significant of a cavity, or of a dilated bronchial tube, it is by no means always present in these conditions. For the perfect production of the sound two factors seem to be necessary—viz. an echoing cavity with free access of air, surrounded by condensed lung tissue. In marked examples, even the whispered voice is plainly conducted (*whispering pectoriloquy*), so that the patient's words are distinctly conveyed up the stethoscope to the ears of the physician.¹ (4) *Ægophony*, yet another modification of vocal resonance, is a term applied to the musical squeak, or 'goat's bleat,' which is heard when there is a slight amount of fluid in the pleura. The voice being compared to a bell, or a musical chord, has certain of its harmonics, especially the graver ones or those of low tension, arrested or obstructed in their transmission through media of varying density (lung tissue, fluid, chest wall). Thus the lower notes of the chord become lost, whilst the higher ones, or those of greater tension, easily penetrate to the auscultator. Hence the voice becomes squeaky and bleating. On the other hand, in cases of large effusion, of pyo-thorax, or of thickened pleura, the vocal resonance becomes completely absorbed, as it were, and is entirely unheard.

c. Cough presents to one's ear divers variations in disease, and it has the advantage of combining certain vocal and respiratory phenomena. It may be cavernous over situations where pectoriloquy and amphoric breathing are heard; or, on the other hand, it may be bronchial where there are bronchial breathing and bronchophony. Beyond this it has a value of its own in physical diagnosis, since by the violent respiratory efforts which it occasions we may elicit râles or other moist sounds which would not be produced by ordinary respiration.

Cough varies in intensity, in character, and in its causation.

¹ A good imitation of bronchophony and of whispering pectoriloquy may be produced by placing one's fingers in both ears and respectively speaking or whispering some test word, such as 'ninety-nine' or the like.

These are discussed or mentioned in the various diseases which it accompanies.

A combined auscultatory and percussion method of detecting pneumo-thorax, or even large cavity, may be practised, by listening over the side of the chest which is presumed to be the seat of pneumo-thorax, whilst an assistant places a coin over another region of the thorax of the same side, and strikes it with some metal instrument. This produces a musical reverberation, which is known as *bruit d'airain*.

(D) MENSURATION of the chest may be made by an ordinary tape measure or by callipers. In comparing the two sides we take the dimensions from the spine of a vertebra to a corresponding level at the mid-sternal line. The callipers are mostly used to record the antero-posterior diameters of the chest. The actual girth of the chest is taken at the line of the nipples, or at the xipho-sternal articulation.

Asymmetry can be graphically recorded by the cyrtometer, an instrument of flat ribbon of lead or other pliable metal, having a hinge which is placed at the back over the vertebral spines. This can then be moulded so as to fit any depression or bulging of the chest wall; and when the instrument is removed its sinuosities are easily traced by a pencil on a sheet of paper.

In a healthy person it should be remembered that measurement of the right side is almost an inch in excess of that of the left.

EXAMINATION OF THE LUNGS

TABLE OF PHYSICAL SIGNS

Normal		Abnormal	
		Condition	Significance
A. Inspection and palpation	Normal movement	Impaired movement	Consolidation of lung ; adhesions of pleura, &c.
		Increased movement	Compensation
		Lost movement	Pneumo-thorax ; fluid in pleura
	Normal fremitus	Jerky . . .	Nervous probably
		Friction fremitus	Pleurisy before or after effusion
		Increased . .	Consolidation of lung
	Normal resonance	Diminished, or Lost . . .	Thickened pleura
		Impaired or diminished	Pleural fluid
		Increased . .	Early consolidation
		Tympanitic .	Emphysema
B. Percussion	Normal dulness	Pneumo-thorax	
		'Skodaic' .	Lung in immediate contact with chest wall by reason of fluid in pleura
		'Cracked pot' (bruit de pot fêlé)	Cavity, or dilated tube
		Diminished .	Slight pneumo-thorax or emphysema
	Normal resistance	Increased . .	Consolidation of lung
		'Tubby' dulness	Fluid in pleura (pus, or serum)
	Normal vesicular murmur	Diminished .	Pneumo-thorax or emphysema
		Increased . .	Consolidated lung, fluid, or new growth
		Harsh, puerile, or exaggerated	Increased compensatory action
		Feeble . . .	Diminished action
C. Auscultation	Normal vesicular murmur	Distant . . .	Collapse of lung with interposition of fluid or other medium
		Prolonged expiratory murmur	Early consolidation ; emphysema
		Crepitation .	Early pneumonia

Normal		Abnormal	
C. Auscultation	Normal vesicular murmur	Condition	Significance
		Crepitation (redux)	Late pneumonia, pleurisy (resolution)
		Bronchial breathing	Consolidation of lungs
		Cavernous or amphoric	Cavity in lung, or bronchiectasis
		Râles (crepitant and subcrepitant)	Breaking down of lung tissue, or fluid secretion in the smaller tubes
		Large moist sounds	Bronchitis of larger tubes
		Gurglings . .	Cavity, or greatly dilated tubes
		Metallic tinkling	Large cavern with surrounding consolidated area
		Friction . .	Pleurisy
		„ (redux)	Pleurisy (after absorption of fluid)
		Succussion splash	Pneumo-thorax, with fluid
		Rhonchus and sibilus	Obstructed bronchial tubes
		<i>Bruit d'airain</i> (a combined auscultatory and percussion sign)	Pneumo-thorax
		Absent . . .	Pus or greatly thickened pleura
D. Vocal	Normal resonance	Bronchophony	Consolidation of lung tissue
		Ægophony .	Pleuritic effusion
		Pectoriloquy	Lung cavity, dilated tube, or consolidated lung
		Amphoric resonance	Large, smooth-walled cavity, or pneumo-thorax (allied to <i>bruit d'airain</i>)
E. Cough	{	Cavernous or amphoric	Large cavity
		Ægophonic .	Pleuritic effusion

The above table is by no means complete; but it is a list of the physical signs most commonly met with, and the nomenclature is that which is in common use. For an extended table see Powell's 'Diseases of Lungs.'

DISEASES OF THE LARYNX AND TRACHEA

LARYNGITIS

Definition.—An inflammation of the mucous membrane of the larynx. Two forms must be recognised, the acute and the chronic.

Pathology.—In a mild attack the laryngeal mirror (which is well tolerated) gives the following appearances : redness and congestion of mucous membrane, especially above and below the true vocal cords. The mucous membrane is also dry and swollen at first, then pours out a thick, gummy, and occasionally a flaky exudation. These changes may involve the whole of the larynx as high up as the epiglottis and aryteno-epiglottic folds ; but the œdema of the true vocal cords is not usually so intense as in other parts. This condition may gradually subside, or the inflammation may become more intense and extend to the sub-mucous tissues and perichondrium, and downwards to the trachea. The muscles of phonation cease to act ; the vocal cords are fixed ; and the exudation, at times, is so firm as to resemble a true membrane. Frequently these changes assume a chronic type, when the mucous follicles become enlarged, and shallow ulcers form. Such conditions are then more than suspicious of a syphilitic or tubercular taint. Their healing process gives rise to much cicatricial tissue, and subsequent laryngeal stenosis.

Acute Laryngitis

Causation.—**DIRECT INJURY.**—Externally, from violent pressure, wounds and blows ; internally, from mechanical

irritation, such as scalding water, acids, inhaled foreign bodies, or from direct inoculation of contagious expectoration. It may also be produced by exposure to cold air, or by shouting and straining the voice. It is frequently secondary to other diseases, notably small-pox, scarlet and other fevers, also syphilis, tubercular disease of the lungs, and Bright's disease. It is said to be more commonly met with in towns than in country places.

Symptoms.—There is usually a little febrile disturbance, and beyond that, such symptoms as would be caused by an obstruction to the entrance of air by the inflamed larynx. Thus there are a slight hoarseness, a husky cough with difficulty of breathing, and dysphagia. From this point the disease may subside, or it may advance to a more severe condition, in which there is almost complete aphonia, with stridulous cough and inspiration, intense pain on movement of the larynx, and a dusky, anxious expression of countenance. The laryngeal secretion, which was at first arrested, becomes profuse and of a muco-purulent or semi-membranous consistence. Added to this there are urgent dyspnœa (shown by the activity of the extraordinary muscles of respiration, staring eyeballs), a feeble irregular pulse, and if relief be not afforded, the patient passes into delirium and coma, death ensuing from asphyxia.

The signs as observed by the laryngoscope are as follows : the larynx is seen to be reddened, especially towards the posterior ends of the vocal cords, which are partially paralysed and thus fail to approximate to each other in the middle line.

The acute attack rarely lasts longer than five days.

Treatment.—**LOCAL.**—Externally apply cold compresses, ice, or hot fomentations. If the strength be good, five or six leeches may be applied, or even a blister ; but these applications are best at some little distance from the seat of inflammation. Internally use atomised drugs, in solution or dry, such as tannate of alum (gr. v. to $\bar{5}$ j.), sulphate of zinc (gr. xx. to oz.), or strong solutions of nitrate of silver (gr. xx. to oz.) or of perchloride of iron. It is essential that local medication be performed by the aid of the laryngoscope, else the drugs may easily be passed into the pharynx and œsophagus.

Should these steps fail to relieve, we may scarify the posterior surface of the epiglottis, or perform tracheotomy. The danger of the latter step is enhanced by its being deferred too long. Nor has it been successful in very young children.

GENERAL.—The patient must be placed in a warm room, the temperature of which should be kept up night and day to a point between 60° and 70° F. Let the atmosphere be moist as well as warm. Use, therefore, a bronchitis kettle, the steam of which may be medicated (eucalyptol, turpentine, or conium). Assistance will be derived from purgatives, diaphoretics; whilst aconite may be tried in the early stages with a view to lowering of temperature. When the acute stage of the attack is passed, saline diaphoretics, and perhaps expectorants (squill, senega, ipecacuanha, and iodide of potassium) are indicated.

It is necessary to be on our guard against a recurrence of the more serious conditions.

Œdematous Laryngitis

This variety of laryngitis is usually acute. It occurs from inhalation of septic matter; or it may be secondary to specific fevers, phthisis, syphilis, Bright's disease, and general inflammation of the areolar tissues of the neck (Ludovic's angina).

Symptoms are mainly characterised by the supervention of rapid and acute dyspnoea, dysphagia, and hoarseness. Laryngoscopically we find extensive œdema of the epiglottis and of the aryteno-epiglottidean folds, the mucous surface generally of the larynx being bright red. The true vocal cords, however, generally escape.

Prognosis will depend entirely on the cause and the amount of respiratory obstruction. Obviously cases due to septic causes are dangerous independent of the amount of dyspnoea.

Treatment—Vigorous local measures are necessary to remove the œdema. Ice may be given to suck. Apply leeches externally over the larynx, or freely scarify the swollen surface of the epiglottis. If relief be not speedily obtained, perform tracheotomy without delay. Subsequent treatment

of the œdema should be by means of astringent sprays or gargles of tannin or catechu.

Chronic Laryngitis

Causation.—A previous acute attack ; chronic Bright's disease ; syphilis ; tubercle ; and new growths. It may also be caused by the constant action of inhaled irritant gas and minute particles suspended in the air ; or it may extend from the pharynx as a result of chronic alcoholism.

Symptoms.—Cough, hoarseness, or complete aphonia, and a constant desire to clear the throat are the principal symptoms, to which may be added, from time to time, some of the signs described in the acute form of laryngitis. Occasionally there is a laryngeal form of ozæna. The laryngoscopic examination, however, is the most important factor in diagnosis. With this instrument we may see a swollen and congested or opaque and granular state of the cords and mucous membrane generally, the aryteno-epiglottidean folds being most involved. The mucous glands are enlarged. In syphilis and tubercular phthisis, ulceration to a more or less extensive degree may be present. Any part of the larynx may be thus eroded, and as a secondary consequence infiltration may extend to the various cartilages of the larynx and to the tissues beyond. Hence the expectoration may be profuse, offensive, purulent, and at times may contain débris of necrosed cartilage and bone. Smart hæmorrhages may thus arise. Recovery is apt to be complicated by stenosis of some one or other part of the tube.

Treatment.—*Local* most important. Treat any acute exacerbation as described in previous section. The laryngoscope should be used in the application of local remedies. Sir M. Mackenzie recommended the application of chloride of zinc (gr. xxx. ad ʒj.). Or we may spray with equal parts of sulphurous acid and water, or use the iodine, creasote, and other astringent and sedative vapours of the Pharmacopœia. Lozenges of catechu, if the mucous secretion be excessive, and of morphia with ippecacuanha if there be much pain or irritation, are valuable.

The *general* treatment will consist of change of climate, sea voyages, cod-liver oil, and tonics in case of plithisical laryngitis; whilst mercury and iodides are indicated in syphilitic disease. Attempts to use the voice should be forbidden. In the treatment of laryngitis, the result of new growth, the question of extirpation thereof (intra-laryngeal, or extra-laryngeal), or the removal of part or the whole of the larynx must be anxiously considered and weighed. It is worse than cruel to delay this surgical help if the necessity be obvious, and the case otherwise suitable.

Aphonia Clericorum (CLERGYMAN'S SORE THROAT)

Definition.—A form of chronic laryngitis, in which there is hypertrophy of the mucous glands of the larynx and at times retention of their contents. The condition is not always confined to the larynx, but may extend to the pharynx and uvula. There is also probably a strong neurotic element in each case.

Symptoms.—Such as would attend a mild form of chronic laryngitis. There is slight hoarseness or huskiness, which is increased during prolonged vocal exertion. The voice after a time may be entirely lost, or reduced to a harsh whisper, or it may suddenly become lost in the middle of a discourse, although fairly strong at the onset.

Treatment.—See Chronic Laryngitis, p. 326.

Tubercular Laryngitis

This variety is almost invariably a secondary complication to pulmonary tuberculous disease.

The **symptoms** are practically the same as in the ordinary forms of chronic laryngitis; and in our experience such cases run an extremely rapid course to a fatal termination. On laryngoscopic inspection we find minute grey tubercles spread over the mucous surface of the larynx. They are most numerous near the arytenoid cartilages, and in the aryteno-epiglottidean folds. Their presence leads to general laryngitis, accompanied by œdema of the mucous membrane.

After a time the tubercular masses ulcerate, the ulcers tending to coalesce, but not to form even then very large patches. In severe and chronic cases the inflammatory processes may extend to the cartilages, and produce erosion and necrosis of these parts.

The **diagnosis** is obviously completed by finding signs of tubercular disease of the lungs.

Treatment.—Apart from the general treatment of the phthisical condition, local remedies are required to allay cough and dysphagia. Morphia and starch insufflations are best. The tube by which they are applied must be passed, by the aid of a mirror, over the epiglottis, and, if possible, directly to the ulcerated surfaces, else in unskilful hands the remedy may be sent into the pharynx. Solutions of hydrochlorate of cocaine, or of nitrate of silver, are also useful.

LARYNGISMUS STRIDULUS (SPASM OF GLOTTIS, FALSE CROUP)

Definition.—Sudden attacks of dyspnœa, due to spasm of the glottis, occurring for the most part in young children during sleep, with intervening periods of apparent health.

Causation.—Obscure. It is said to ‘run in families,’ and has been attributed to : (i) irritation of the recurrent laryngeal nerve from pressure of enlarged bronchial glands or of thymus ; (ii) irritation of vagus centre ; (iii) rickets ; (iv) reflex irritation from worms or irritant food in the intestines ; (v) direct irritation of the mucous membrane of the larynx from cold air, &c.

Pathology.—The laryngoscope reveals an injected mucous membrane with œdema below the vocal cords. There is at first a dryness of the mucous surfaces, which is followed by copious catarrhal secretion.

Symptoms.—The disease begins suddenly, almost always in the night, and usually follows a slight ‘cold.’ The child is roused from sleep by a fit of coughing of a ‘croupy’ character. The inspirations are laboured and noisy, attended by dilatation of the nostrils, and a characteristic whimper or cry between

the inspiratory efforts. The expression of face is anxious, the skin hot and flushed, and the pulse quickened. The struggle for breath is so severe that the child may tear at its mouth or throat, or cling to its nurse for help. The attack usually lasts from about twenty minutes to an hour, when the patient sinks into a quiet sleep with few or no symptoms of the previous struggle beyond a little inspiratory stridor. A second attack, however, may occur spontaneously during the night; or it may be immediately excited by pressure on the windpipe. Sometimes a similar outbreak occurs during the next one or two succeeding nights, but rarely beyond this, although renewed attacks may supervene during a twelvemonth.

Treatment.—There is no remedy against the recurrence of attacks. Small doses of iodide of potassium and aconite have been advocated.¹ Emetics cause unnecessary weakness. A ‘whiff’ of chloroform is probably more useful. A greased rag applied to the throat is a soothing remedy. Small doses of oxymel of squill may be given during the daytime. Protect the child against exposure to cold. Let the diet be carefully regulated and restricted to milk during the periods of attack. Keep the patient in a warm room till all symptoms have vanished. The disease is seldom fatal, but it may precede an attack of true membranous croup. (See Henoch’s ‘Children’s Diseases,’ New Sydenham Society, 1889.)

TRACHEITIS

Definition.—An inflammatory condition of the mucous membrane of the trachea. It may assume an acute or a chronic form.

Causation.—Similar to laryngitis. It is also frequently secondary to inflammatory affections of the large bronchi, or it may be caused by pressure (aneurysm, new growth), injury, surgical operations, *e.g.* tracheotomy, &c.

Treatment.—As in laryngitis.

¹ ℞. Potass: Iodidi gr. j.; Tinct: Aconiti mīj.; Syrupi Scillæ mx. Aquam ad ʒj.

DISEASES OF THE BRONCHI

BRONCHITIS

Definition.—A catarrhal inflammation of the mucous membrane of part or of the whole of the bronchial tree.

The affection may be acute or chronic, and it may be limited to the larger tubes or extend to the smaller ramifications.

Acute Bronchitis

Causation.—*Age.*—No age is exempt. *Sex.*—Males suffer more than females, on account of more frequent exposure to climatic variations. *Climate and Season.*—It is endemic in northern latitudes, and most prevalent in winter and early spring. *Occupation.*—It is predisposed to by occupations which involve exposure to cold air (coachmen, riverside workers, &c.). The direct cause is the inhalation of cold, damp atmosphere, or of irritating dust or chemical matters suspended therein, as well as poisonous gases, fogs, &c. There may also be some defect in the naso-pharyngeal mucous membrane, which necessitates oral respiration.

It is also at times secondary to specific fevers, to gout, Bright's disease, and chronic alcoholism; or to intra-thoracic pressure on the bronchi (aneurysm, new growth, gumma); or to septic abscesses or cavities in the lungs. It not infrequently complicates obesity. A previous attack is also a predisposing causal factor.

Pathology.—The extent of the pathological changes in the bronchial mucous membrane and peribronchial tissues will depend entirely on the intensity of the attack. In the early

stages the mucous membrane is red and congested, the capillary plexus supplying it being engorged with blood or presenting minute hæmorrhages. The mucous glands are obstructed, and their secretion consequently arrested. The membrane is thus dry and swollen, and the calibre of the tube diminished. Subsequently the mucous glands pour out a watery fluid, which becomes mixed with air-bubbles and possibly blood. The epithelial cells become detached and mixed with the watery secretion, and are replaced by leucocytes and embryonic cells. At a later stage the secretion becomes muco-purulent and finally purulent, containing fatty, granular, and degenerated cells, which constitute the bulk of the expectoration.

If the inflammatory process extend to the submucous and peribronchial structures, there is proliferation of the cellular elements of the connective tissues; the muscular fibres may be irritated to excessive spasmodic action, or they may atrophy and degenerate. Usually the lymph spaces are crowded with leucocytes.

Ulceration of the bronchial tubes is comparatively rare, but it may take place. When it does occur, there is necrosis of the cartilaginous rings, and subsequent excavation of the bronchial passages into irregular yet well-defined cavities, which are only kept from communication with the neighbouring air-cells by a zone of inflammatory tissue.

If the affection extend to the smaller tubes, the pathological processes are the same, but the results are more disastrous, inasmuch as the inflammatory exudation has a greater tendency to cause complete obstruction to the narrowed air-passages, with subsequent collapse of the lung tissue immediately behind the constriction. On the other hand, especially if the affection be of a chronic nature, the infundibula leading from obstructed bronchi become greatly distended by retained air, and a form of emphysema may result before this air has had time to become absorbed.

It should be noted that the post-mortem naked-eye appearances of acute bronchitis are by no means always so marked as the physical signs before death would lead us to expect.

Symptoms.—At the onset the symptoms are those commonly associated with ‘a cold.’ The patient often complains of sore throat, nasal discharge, and coryza. This is followed by quickened pulse, thirst, loss of appetite, and general febrility; the symptoms of fever being more pronounced in children.

Then follow symptoms of obstruction to the respiratory function. There is incessant cough, with pain and discomfort in the chest, most marked over the manubrium. The breathing is quickened (thirty in a minute), the face congested and livid, whilst the pulse, though somewhat accelerated, is full and soft. The patient, as a rule, can lie in any position, unless the capillary tubes are extensively inflamed, when he finds least distress by lying on his back with the shoulders raised. Congestive headache is a common accompaniment. All these adverse symptoms are aggravated at night time. About the second or third day the tubes pour out their secretion, and much of the discomfort passes away. The expectoration is at first watery, then glairy and tenacious, and finally becomes muco-purulent or purulent.

After the lapse of a week or ten days, if the disease be limited to the larger divisions of the bronchial tubes, the dyspnœa subsides, the cough diminishes, and expectoration is lessened and more easily raised, and the patient is gradually restored to health. On the other hand, inflammation of the smaller pipes is marked by more urgent dyspnœa, more frequent cough, greater difficulty of expectoration, and a tendency to speedy death from asphyxia.

Physical Signs.—Always well-marked. The respiratory movements of the chest are quickened and exaggerated, especially in the superior costal regions. Percussion is unimpaired, unless there be large tracts of collapsed lung. In the dry stage, auscultation detects sonorous rhonchi in the larger, and sibili in the smaller tubes; whilst later on, when the mucous secretions are more plentiful, these sounds give place to crepitant râles, varying in pitch and intensity according to the size of the tubes in which they are produced.

The moist sounds are heard equally on both sides, and are

most abundant at the bases of the lungs. These sounds, both moist and dry, may be so loud as to be heard by the patient himself or by his attendant. And further, most cases of bronchitis present the mixed sounds: some tubes yielding a dry, sonorous note; some being sibilant and musical; whilst others, again, often produce râles and other moist sounds. This merely shows that the tubes in different parts of the bronchial tree are in different stages of the disease, and therefore under different conditions.

Diagnosis.—Acute bronchitis may be confounded with (1) acute phthisis, and (2) œdema of the lungs. (1) The former condition may be suspected if the patient be a young adult with high temperature, and dyspnoea in excess of the physical signs, and when auscultation detects patches of tubular breathing with ‘clicks’ and larger moist sounds. Further, the physical signs would probably be more accentuated in the apices and upper lobes. Microscopic examination of the stained sputa would also reveal the presence of the tubercle bacillus. (2) If œdema of the lungs be suspected, we should probably find a dull, resistant percussion note over the bases, together with signs of feeble heart-wall or of valve lesion.

Prognosis.—Favourable as a rule. It is more fatal in infants and in the aged. The prognosis is also rendered more grave if the inflammation extend to the smaller tubes and air-sacs, or if the disease occur in patients already debilitated by fevers, chronic heart disease, or other exhausting illness.

Treatment.—The patient should be restricted to bed. The atmosphere of the room should be maintained at a warm temperature (65° F.) by free use of a steam kettle. If seen in the early stages, give saline purgatives with small doses of opium (Dover’s powder, gr. iv). In severe attacks, antimony, in small repeated doses, is of service. When the attack is well-established iodide of potassium with alkalis will render the secretion less viscid and promote expectoration. Bleeding by leeches, or cupping glasses to the chest, or strong counter irritation (blisters, sinapism) may be employed with advantage in robust patients, or when there is manifest embarrassment of the right side of the heart. Linseed poultices

are objectionable on account of their weight and other discomforts. When the bronchial secretion becomes free and muco-purulent, assist expectoration by ipecacuanha, squill, senega, liq. ammoniæ acetatis.¹ For this purpose also emetics are more useful in children than in adults. A pill of purified tar, Dover's powder, and benzoin, three times a day, will often give immense relief by allaying cough and promoting expulsion of sputa. Opium, henbane, chloral hydrate, are extremely useful as sedatives; but their effect upon respiration and circulation must be carefully watched. A dusky skin, or a tendency to delirium would caution us against their use. Occasionally the diluted fumes of sulphurous acid give relief, and it is astonishing how well the patient bears this remedy.

If the disease tends to chronicity, still continue the stimulant expectorants. Free doses of alcohol are then often of great service. The most useful treatment, however, is medicated inhalations, such as eucalyptus, creasote, iodine, or a spray of vinum ipecacuanhæ. The diet throughout should consist of milk, farinaceous foods, eggs, and meat broths.

During recovery tonics are essential, together with warm clothing, so as to obviate a recurrence of the attack.

Chronic Bronchitis

Causation.—A previous acute attack. Constant exposure to dust or irritating gases. It may also be a complication of gout, alcoholism, Bright's disease, &c.

Symptoms.—Vary, according to the intensity of the disease. A mild attack may occur each winter, and pass off during the summer, and only be attended by some slight dyspnoea, cough, and the expectoration of small, dusky, tough pellets of mucus.

On the other hand, symptoms may be as severe as in the acute variety, on account of the already damaged bronchial mucous membrane.

In the typical, well-marked case there is dyspnoea and cough,

¹ R. Vini Ipecac: ℥x.; Tinct: Scillæ ℥xv.; Liq: Ammon: Acet: ℥xxx.; Infusi Senegæ ad ʒj. Miscæ.

with viscid, scanty expectoration. Or the sputa may be purulent, mixed with ropy mucus and occasionally blood, denoting a recent acute inflammatory state occupying some special locality. The cough is often worst and the expectoration most profuse in the morning or on lying down. The patient also complains of a sense of tightness of the chest or between the shoulders, and also of pain and tenderness in the lower costal regions, probably the result of muscular strain from coughing. The pulse is of increased frequency, and there is some slight fever. The respiration is not only laboured, but is generally attended by an audible whistling or sibilus during expiration, and the expiratory flow of air is so noisy as to be distinctly heard whilst the patient is speaking. There is also a distinct 'facies' difficult to describe, but easily detected by an experienced observer; its principal characters are a dusky aspect of countenance, congested conjunctivæ, and somewhat widened palpebral fissures, giving the patient an anxious and somewhat melancholy look. Dyspepsia is a common accompaniment. These conditions may continue for some years with remissions and exacerbations; or the patient may lapse into a cachectic state with increased cough, wasting, sweating, hectic fever, and symptoms resembling phthisis.

Complications.—If the disease be of long standing, pulmonary emphysema of the lungs supervenes, with the physical signs common thereto, and constant dyspnœa. The chest becomes barrel-shaped and hyper-resonant; the liver and spleen are displaced downwards; the heart's dulness is overlapped, and therefore diminished in area; there is marked epigastric pulsation from hypertrophy of the right ventricle; and the extraordinary muscles of respiration are more developed than is natural.

The bronchial tubes may become dilated (*bronchiectasis*) with, in some cases, extremely fetid expectoration due to the presence of butyric acid. In others the sputa may be extremely profuse (*bronchorrhœa*), or they may contain portions of necrosed cartilage or fibrous tissue.

Further complications, due to obstruction of the flow of blood from the right heart (*passive congestion*), generally

terminate the disease. There are then observed hypertrophy of the right ventricle, possibly a tricuspid regurgitant murmur, dilatation of the right auricle, an enlarged, swollen (*nutmeg*) liver, albuminuria, and anasarca. These dropsical conditions are more frequent in chronic bronchitis than in phthisis, owing to the cough being more severe, and the prevalence of the disease in aged people, when the vessels are weakened by decay.

Death is usually due to asphyxia or exhaustion.

Varieties.—1. A PLASTIC FORM of chronic bronchitis is occasionally met with in which the expelled secretion, when teased out in water, is found to consist of a complete mould of some of the larger tubes and their offshoots.

2. A GELATINOUS OR DRY FORM, common in London and smoky towns, characterised by little or no cough, but by hawking or clearing of the throat, by which a small sago-like grey pellet is expelled. The lumps, which are at times hard to expel, are heavy, gelatinous-looking, elastic masses, and appear to have been formed in the larger bronchi. Microscopically they consist of large columnar or spherical epithelial cells containing black pigment (smoke). This form is attended by little or no discomfort, except when complicated by acute exacerbations. It is, however, frequently attended by distressing and even violent cough, a cough which is the more exaggerated by the patient's irritable efforts to expel the plug of mucus which is not yet detached.

Treatment.—Palliative only, when the chronic form is well-established. If possible, remove the patient to a warm climate during winter. Sea-side resorts and elevated localities are not, as a rule, so suitable as warm, moist, and sheltered places. The patient's clothing should be warm, yet light. He should, if possible, give up any occupation which causes bronchial irritation. The diet should be light, nutritious, and in sparing quantity. Certain articles of diet, such as cheese, pastry, &c., seem to exert a baneful influence by reason of their indigestibility, and should therefore be forbidden; similarly large meals with long intervals between, and notably heavy suppers, must be forbidden. Experience shows, however, that

onions may with advantage be freely added to the dietary, often with great relief. This vegetable seems to act as an expectorant, an antispasmodic, and a sedative.

The medicinal treatment should be directed mainly to alleviate symptoms. The so-called 'lowering' treatment is not recommended. To assist expectoration order inhalations of steam, to which may be added tar, creasote, eucalyptus oil, or carbolic acid. Stimulating expectorants (senega, squill, ammonia) are of advantage to debilitated subjects, as in the later stages of acute bronchitis.¹ Dyspnoea, especially if due to fresh inflammatory trouble, may be met by blistering liniments of cantharides, croton oil, turpentine, or, if it be associated with arrest of secretion, alkalies with iodides are of great service. The different ethereal preparations may also from time to time be added if there is much bronchial spasm. Relief is frequently obtained from nightly inunction of the chest, especially the sides, with some greasy liniment (Russian tallow, cod-liver oil, &c.).

For the cough give hyoscyamus, tolu, or compound tincture of camphor in small doses; whilst if it be attended by difficulty of expectoration, the gum ammoniacum may be added. For *bronchorrhœa* the oleo-resins of cubebs or copaiba are of great service if their nauseous taste can be tolerated.²

Bronchiectasis

Definition.—Dilatation of bronchial tubes.

Causation.—Almost invariably secondary to some other disease. It may be caused by the obstruction within the larger bronchial tubes in chronic bronchitis, or by pressure from without by aneurysm or new growth; or from fixation of the pulmonary tissue by pleuritic adhesions and fibroid phthisis, the dilatation being brought about by increased inspiratory effort; or it may be due to condensation of adventitious fibrous growth keeping the tubes permanently over-dilated.

¹ ℞. Ammon: Carb: gr. iv.; Potass: Iodidi gr. iij.; Potass: Bicarb: gr. x.; Tinct: Scillæ mxx.; Infusi Senegæ ad ʒj. Misce.

² ℞. Bal: Copaibæ mxx.; Liq: Potassæ mxx.; Ol: Cubebæ ʒj.; Spt: Chloroformi mxx.; Aquæ Menth: Pip: ad ʒj. Misce.

Finally, it may result from softening and destruction of the histological elements of the bronchial tubes themselves.

Pathology.—There are three different varieties of dilatation: 1. the FUSIFORM; 2. the GLOBULAR, or SACCIFORM; 3. the IRREGULAR. The first variety is generally met with in the larger tubes; the second occurs in the terminal air-passages; whilst the third is often associated with excavation of neighbouring lung tissue. There is invariably precedent inflammatory change in the bronchial passages, with probably some necrosis of their histological elements, the dilatation subsequently being effected either by increased respiratory efforts and cough, or by cicatricial contraction of thickened inflammatory tissue.

Symptoms.—The symptoms, on the whole, much resemble those of phthisis. There is cough, some fever, wasting, and much muco-purulent expectoration, which is usually disgustingly foetid. The dilated tubes appear to empty themselves periodically, after some favourable change of position. The sputa contain no bacilli. Hæmoptysis, however, is not frequent except in the last stages; but cyanosis, clubbed fingers, and other symptoms of embarrassment of the right heart are common.

The physical signs are such as would exist in lung cavities. There will be found cavernous respiration, whispering pectoriloquy or bronchophony, and coarse moist sounds. It should be remembered that the bases of the lungs may be the only regions affected.

Treatment.—Palliative only. Iodide of potassium in small continued doses may have a beneficial effect. Relieve the cough by opium and its derivatives. The extreme factor of breath should be treated with medicated vapours (tar, eucalyptus, creasote). Give nutritious food, and cod-liver oil to combat the progressive emaciation. Aspiration and anti-septic drainage of localised cavities have been practised.

ASTHMA

Definition.—Dyspnœa due to spasm of the muscular fibres of the smaller bronchial tubes, occurring in paroxysms with intermissions of good health between.

Causation.—*Age.*—No age exempt, but it is more frequent in middle and advanced life. *Sex.*—Men suffer more than women. *Climate.*—A disease of cold, damp countries; or where high winds are prevalent. *Heredity* is a strangely marked predisposing cause, especially in the purer and uncomplicated forms. *Reflex.*—It may originate by reflex irritation, from indigestible food, a loaded state of stomach or bowels, cold to the surface, or from irritating matters circulating in blood (hæmic asthma), such as occurs in gout, rheumatism, &c. *Direct irritation.*—From impure air or other inhaled irritant, *e.g.* dust, smoke, ipecacuanha powder, pollen of flowers, &c. (specific asthma). In many instances it is a manifestation of a *neurotic habit*. Thus it is associated with epilepsy in one patient, with angina in another; and in most instances the attack itself is excited by some severe nervous disturbance or by sexual excesses.

Pathology.—No marked post-mortem changes so far as the respiratory apparatus is concerned. The general view is that there is tonic spasm of the bronchial muscles, or a vascular swelling of the mucous membrane of the tubes.

Symptoms.—An attack may be foretold by restlessness, irritability, itching of some part of skin, nose, or eyelids, or by dyspepsia; but usually the seizure comes on suddenly at any hour, though most frequently in early morning. The patient often awakes from sleep with a sense of oppression and tightness about the chest. The chest is apparently fixed in expansion; the patient grasps the furniture or other object in order to bring the extraordinary muscles of respiration into play; he desires to sit at an open window to obtain air. The speech is short and husky; the eyes are prominent; the face is dusky, or livid, and covered with cold sweat; the pulse is small; the urine pale and of low specific gravity. After an

hour or two of agonising dyspnœa the spasm relaxes, breathing is easier, and the attack subsides with the expectoration of frothy mucus or tough pellets not unlike boiled sago. The expectoration also often contains spirally twisted threads of mucus (Curschmann's spirals). Hæmoptysis is rare. An attack may be suddenly induced, and also as suddenly checked, by severe mental emotion, such as fright, joy, &c.

Physical Signs.—The percussion note is usually hyper-resonant; the respirations are slow, with marked prolongation of the expiratory murmur. Auscultation reveals scattered dry sounds, rhonchi especially; moist sounds are rare.

Diagnosis.—A somewhat similar condition may be produced by organic disease of the brain; pressure on the vagus nerve or its recurrent branch; pressure by bronchial glands; abductor paralysis of vocal cords; chronic disease of the heart or aorta; Bright's disease. The diagnosis in each of these conditions will be assisted by careful investigation of the nervous system, by exploration of the chest, by laryngoscopic examination, together with the absence of the characteristic signs of asthma. In bronchitis, attacks of dyspnœa are not sudden in their invasion, whilst the dyspnœa of emphysema continues and is not paroxysmal.

Prognosis.—Usually good. Patients live to a good age, probably on account of the care they take of themselves. They generally die of heart disease or other complication.

Treatment.—**PROPHYLACTIC.**—The patient should select a locality in which the climate and the atmospheric conditions have been proved to suit him. No further rule can be laid down, some patients thriving best in smoky towns, others in pure elevated atmospheres; relaxing climates are best on the whole. The diet should be spare; the meals light, digestible, and not at too long intervals. Suppers should be avoided. Correct any disorder or overloading of stomach and bowels. Nervine tonics (phosphorus, arsenic, quinine) may be given between the intervals of attacks.

PALLIATIVE.—During an attack we may bleed if there be signs of much congestion or of high arterial tension. An emetic is indicated where the stomach is overloaded. Citrate

of caffein appears to be the best remedy.¹ Coffee, hot, strong, and pure, frequently gives relief. Other remedies acting speedily are, inhalation of amyl nitrite, chloroform, 'Himrod's cure,' 'Senior's remedy,' 'cigarettes de Joy,' stramonium cigarettes. The subcutaneous injection of sulphate of atropine ($\frac{1}{200}$ gr. cautiously increased) often gives good results. Dr. C. Theodore Williams advocates the compressed air bath. If there are signs of chronic bronchial changes give iodide of potassium in combination with bromide of ammonium and the ethereal tincture of lobelia. Patients are also frequently relieved by rubbing the sides of the chest and the epigastrium with oil or some simple liniment.

HAY ASTHMA (HAY FEVER)

Definition.—A disease supposed to be due to stimulation of the mucous membrane of the nose and Schneiderian membrane by dust of hay, pollen of flowers, especially roses, and irritating vapours, accompanied by much sneezing, lachrymation, and nasal discharge.

Causation.—Both sexes suffer, but it is more prevalent in males. The disease is generally epidemic in May, June, and July, during the period of efflorescence of plants. It is more frequent in the better classes; and, seeing that farm labourers and other workers in the fields rarely contract the disease, there would appear to be some other causation than hay dust. Possibly, as in many other cases, some peculiar idiosyncrasy acts as an exciting cause.

Pathology.—Nothing is definitely known. Mechanical irritation by fine dust is the probable exciting cause, which may produce temporary or permanent thickening of the mucous membrane covering the turbinate bones, and also of the bronchial tubes. Some authorities insist that there is always a predisposing condition in the mucous membranes of the naso-respiratory tract in the form of chronic rhinitis.

Symptoms.—The disease commences with itching of nose,

¹ R. Caffeinæ Citratis gr. ij.; Ext: Stramonii; Ext: Hyoseyami, āā gr. ij. Misce; ft. pil.

a sense of oppression and pain under the manubrium sterni, and frontal headache ; then follow lachrymation, sneezing, a dry, hacking cough, and symptoms of naso-bronchial catarrh, together with attacks of dyspnœa, which are distinctly paroxysmal. The end of an attack is accompanied by a thin, clear discharge from the nose and respiratory tract, marked bodily prostration, and mental distress. The eyelids in consequence of the constant lachrymation are puffed and swollen, and frontal neuralgia is a frequent accompaniment.

Treatment.—Immediate removal from source of irritation. The seaside is the best resort. All sorts of remedies have been advocated. Our best results have been obtained by constant irrigation of nostrils with warm water, with or without the addition of bicarbonate of soda (gr. x. ad $\bar{5}$ j.); or a weak solution (five per cent.) of hydrochlorate of cocain, may be substituted. As regards drugs, iodide of potassium gives in some cases distinct and speedy relief. The hypertrophic condition of the nasal mucous membrane may require the galvano-cautery for its cure. (See Sir Morell Mackenzie on Hay Fever.)

DISEASES OF THE LUNGS

CONGESTION OF THE LUNGS ¹

Definition.—An excess of blood in the capillaries of the lungs.

Causation.—It may exist as the early stage of pneumonia, or as the result of prolonged exertion or violent respiratory efforts (*active* congestion). It occurs also in the course of severe febrile and debilitating diseases (*hypostatic* congestion). Finally, it may result from mechanical obstruction to the flow of blood through the heart, whether it be caused by stenosis of orifices, defect of valves, or debility of ventricular muscle (*mechanical* congestion).

Pathology.—The lung at the site of the congestion is abnormally red, owing to the capillaries being gorged with blood, whilst the air-cells are diminished in size on account of the dilated capillaries. On section much blood-stained serous fluid exudes. Still the lung remains for the most part crepitant. After long-standing congestion the part affected is tough, dark coloured, and contains very little air. The congestion may also extend to neighbouring bronchial tubes. Circumscribed hæmorrhages (*pulmonary apoplexy*) are not uncommon.

Symptoms are mainly those of dyspnoea, and lividity, with cough and attendant expectoration of glairy, blood-stained serum, or severe hæmoptysis. To these would be added, when large tracts of lung tissue are involved, dulness, increased vocal phenomena, and subcrepitant râles.

¹ The term is often used carelessly, and by the public is accounted a condition of great gravity.

Treatment should be mainly directed to the exciting cause. Rest is essential. Try to restore the balance of circulation by hot fomentations, blisters, or local blood-letting. Digitalis is especially indicated in cases of cardiac failure. In debilitated subjects rely on nourishing foods and stimulants (alcohol, ether, ammonia, &c.).

ŒDEMA OF THE LUNGS

Definition.—Exudation of serum into the lung tissue (*dropsy* of the lungs).

Causation.—Bright's disease, purpura, anæmia, scarlet fever, and indeed any disease or morbid condition of the blood and circulatory apparatus which predisposes to general dropsy. It is therefore nearly always a secondary lesion.

Pathology.—The parenchyma of the lungs is infiltrated with colourless serum, the organs themselves being sodden, swollen, and indented by ribs and other resistant structures. They are pale in colour, less crepitant than normal, pit on pressure, and on section freely exude clear or blood-stained serum, in which some air-vesicles are entangled.

Symptoms and Physical Signs.—Usually of a chronic type.¹ They consist mostly of dyspnoea, increased frequency of respiration, cough, with expectoration of clear watery or frothy serum. There is no fever. The percussion note is dull on *both* sides, especially towards the bases. The respiratory murmur is feeble, and often attended with crepitant râles. As the condition is a local manifestation of general dropsy, we should expect to find œdema in other situations.

Treatment.—Rest in bed is essential. Beyond this it is necessary to adopt measures to relieve the dropsy locally by dry cupping. If the kidneys be primarily at fault, give mild diuretics, diaphoretics, and watery purges till the physical signs be improved. If the condition be dependent on heart disease, digitalis, strophanthus, with ether and other stimulants, are indicated ; to be supplemented, when improvement takes

¹ An acute œdema has been described by various authors, the causation and pathology of which are not yet clearly determined.

place, by iron, tincture of nux vomica, and other tonics. The diet should be nutritious and stimulating.

HÆMORRHAGE (HÆMOPTYSIS)

Definition.—Spitting of blood emanating from the lungs or respiratory passages. It is a symptom rather than a disease.

Causation.—The hæmorrhage may come (*a*) from the MUCOUS MEMBRANE OF THE WINDPIPE OR OF THE BRONCHIAL TUBES (*bronchial hæmoptysis*); (*b*) from the CAPILLARIES OF THE AIR-CELLS (*pulmonary hæmorrhage, pulmonary apoplexy*).

(*a*) In the former category may be placed bronchitis, ulcerations of trachea, larynx, bronchial tubes, whether from phthisis, new growth, syphilis, or diphtheria. It may occur also in hæmophilia and in persons of feeble health. The hæmorrhage may arise also from extension of aneurysm, abscess, or hydatid cyst into the air-passages.

(*b*) PULMONARY HÆMORRHAGE varies from the slight amount seen in croupous pneumonia to the alarming extent often present in advanced cases of phthisis and heart disease. Between these degrees we find it due to feeble circulation, Bright's disease, purpura, embolism of the pulmonary artery. It may supervene on severe exertion, especially in high altitudes; and is not infrequently hereditary between the ages of fifteen and thirty, with no physical cause to be detected. We have never been satisfied that it exists vicariously to menstruation.

Symptoms.—Hæmorrhage may come on with or without warning. Often, however, there is a precedent cough, followed by a tickling in the windpipe, a sense of irritation under the sternum, and a salt taste of blood. This causes an effort of expectoration, when blood is expelled; subsequent cough increases the quantity. Or the mouth may be suddenly filled with a large quantity of blood, without any cough or premonition. Generally the blood is of bright colour, and frothy from the admixture of air; but it may be dark, or moulded in pellets, according to the length of time it has escaped from

the vessels. Generally after the onset of hæmoptysis there is a subsidence, followed by a return in a few days.

Beyond these symptoms the patient's pulse is quickened, the face blanched and perhaps bathed in cold perspiration, and he has a tendency to faintness ; but the temperature is raised. These symptoms, however, are, as a rule, due to alarm and nervous shock, rather than to the loss of blood.

Physical Signs.—Auscultation of the chest will probably reveal presence of disease of lungs or of arterial system, though not always, even when bleeding comes from the respiratory passages. But prolonged examination of chest must on no account be made, as it may increase the bleeding ; and it is useless, since an accurate diagnosis of the state of lungs and heart can only be made after hæmorrhage has ceased. In some cases in which it occurs after some severe exertion (rowing, wrestling, &c.) there may be no physical signs whatever.

Ascertain that the blood does not come from the nose, fauces, or mouth ; if from the former it may be seen trickling down the posterior wall of the pharynx when the head is reclining on a pillow, or it may escape from the anterior nares if the head be thrown forward. If it come from the stomach the blood is dark coloured, partially digested, and mingled with mucus and food ; further, it has been vomited up with antecedent nausea.

Prognosis.—This will obviously vary according to the cause and the amount of the hæmorrhage, the history and condition of the patient. Some bear losses so much better than others. The dangers are : (a) IMMEDIATE ; (b) REMOTE.

(a) IMMEDIATE.—There may be fatal syncope, although only small quantities of blood are lost : here it is due to alarm. Asphyxia may occur in debilitated subjects, owing to their inability to expectorate the blood as it flows.

(b) REMOTE.—Gradual sinking from loss of blood ; occurrence of pneumonia as a sequel to the hæmorrhage, or from the application of ice to the chest in weakly patients ; it frequently accelerates phthisical disease when occurring in those already slightly affected.

Treatment.—Rest alone is sufficient in mild cases. The patient's room should be cool; he should be prohibited from speaking, and encouraged to restrain the cough as much as possible. Sudden movements of the body are also to be deprecated. The food should be light, unstimulating, and cold. He may have, in addition, cold water to drink, and ice to suck. Ice-bags to the chest, over the presumed site of hæmorrhage, are probably worse than useless. Nor are we in favour of astringent drugs by the mouth. Sulphuric acid, gallic acid, acetate of lead, turpentine, have their advocates, but it is probable that by causing constipation they add to the blood pressure. Keeping in view the alarm of the patient and the panic of the household, it will be found that opium by the mouth, or morphia subcutaneously, is the best remedy. Give the drug to its full physiological effects. The calm and allayed fear produced by opium will often be succeeded by arrest of the bleeding. Ergot may be added if the hæmorrhage be severe, and digitalis may be combined advantageously if the pulse be rapid.¹ (See 'Lancet,' vol. i. 1884.) If there be much depression turpentine (m.x.), with or without alcohol, is often successful.

ACUTE PNEUMONIA

Definition.—An acute inflammation of the parenchyma of the lung producing consolidation. Two forms of pneumonia are recognised: (*a*) CROUPOUS PNEUMONIA, involving a lobe or large tract of lung tissue; (*b*) CATARRHAL or LOBULAR PNEUMONIA, which occurs in isolated patches, and is usually a secondary affection.

(*a*) Croupous Pneumonia (LOBAR PNEUMONIA)

Causation.—No age is exempt; the disease probably most frequent in early adolescents. *Sex.*—Males suffer more frequently, owing to their greater exposure. *Season and Climate.* The disease is especially prevalent in winter and spring, and

¹ R̄. Tinct: Op̄ii m̄v.; Tinct: Digitalis m̄x.; Ext: Ergotæ Liq: m̄xv. Aquam ad ʒj. Misc.

in cold and in temperate climates, where it frequently occurs in epidemics. Localised epidemics in unsanitary localities or houses are not uncommon ('Lancet,' July 1888). *Other Diseases*.—As predisposing causes we may mention debilitating diseases and cachexia, also the effects of alcohol, syphilis, plethora. It also appears to be in some way connected with acute rheumatism. It may be a complication secondary to inflammation of the bronchial tubes or pleura; it may also occur from the effects of cardiac emboli, or the passive congestion following valvular disease of the heart.

An attack seems to predispose to a subsequent seizure, but a third is rare.

Pathology.—A typical example of inflamed tissue. The disease runs through three well-marked stages, viz. :—

1. DETERMINATION OF BLOOD, OR ENGORGEMENT.—The capillaries are tortuous, and choked with blood corpuscles. They exude a viscid, albuminous fluid. There is proliferation of the cells lining the alveoli, and also of the connective-tissue cells. The lung during this stage is red, contains some air, floats in water, and is extremely friable. 2. RED HEPATISATION.—The lung now becomes solid, it contains no air, and sinks in water. The colour on section is a brown red tint. The lung tissue is still friable, and granular on section, owing to the alveoli being overdistended with the exudation of viscid serum and fibrin, thus choking them with a semi-solid mass, which consists of migratory leucocytes, red blood-cells, and the alveolar epithelium, all being held together by a fibrillated fibrinous network. 3. GREY HEPATISATION.—The inflamed tissue gradually changes colour, becoming a mottled, dirty white tint, this being due to a diminished supply of fresh blood, and also to degenerative changes in the leucocytes and exuded corpuscles (pus). The mould of inflammatory products begins to shrink and come away from the alveolar walls. This is followed by fatty and degenerative changes in the proliferated cellular elements, which finally liquefy and become converted into pus. The tissue is still friable and granular, and also sinks in water. The affected area is easily distinguished from the rest of the lung by its being altogether swollen and in-

creased in size. In all cases where pneumonia is near the surface of the lung it is attended by dry pleurisy. The affection is more frequently found involving the right lung than the left, and the bases rather than any other portion.

From this stage (i) *resolution* may occur: the contents of the alveoli being partly absorbed by the lymphatic system, and partly expectorated; whilst the lung tissue gradually resumes its normal character, but with, for a time, impaired elasticity. Or (ii) the *inflammatory process* may proceed to the actual destruction of lung tissue and the formation of abscess or the supervention of gangrene.

According to Friedländer, Dreschfeld, Klein, and others, there are found in sections of pneumonic lung specific micro-organisms of oval shape (pneumococci), the presence of which appears, at least in some cases, to be the cause of the disease. Pneumonia should therefore, if this view be correct, be considered as a specific inflammation. In confirmation of this, from a clinical aspect, Dr. Ord has for some years contended that pneumonia may be regarded as a condition akin to erysipelas.

Symptoms.—The onset is sudden, and marked by rigors, hurried respiration, fever, cough, and pain over the affected part. Such symptoms, without physical signs, are not diagnostic; they merely denote outlying inflammatory states or catarrhs. Murchison taught that there are four diagnostic signs, viz. lividity, dyspnoea, rusty sputum, and herpes labialis.

The initial stage is characterised by rigors, or shivering, and a general sense of febrility. It may be ushered in by convulsions in children. Dyspnoea is an early symptom. It is more or less marked according to the amount of lung tissue involved; it is an especial feature in apical affection. The respirations are increased from twenty-five to forty per minute, but are regular; the chest movements being limited on the affected side. With a view to lessen the dyspnoea the patient reclines on his back with his shoulders raised. The temperature, which rose suddenly at the onset of the disease (102°), mounts higher when the inflammation is well-established, and remains high (104.5°) during the whole period of the illness.

Generally it is highest about the third or fourth day, and is seldom above the range indicated, unless there is some under-current malady (e.g. pyæmia, erysipelas, &c.). When the apex is involved, however, a temperature of 105° , or even 107° , has been observed. Cough is always present, and is attended by expectoration, which varies in tint and consistence in the different stages. Thus in the first days of the attack it is viscid, tenacious, and full of minute air-bells; then it becomes characteristically rusty, or even prune-juice coloured, the blood being intimately mingled or churned up with the secretion from the alveoli. Subsequently the phlegm becomes heavier and purulent, especially when the minute bronchioles become involved. Pain over the site of the inflamed lung is generally of a heavy, burning character, but not so sharp and lancinating as in pleurisy, unless, as often happens, the pleura itself be involved. Beyond the above we find also other marked signs. The pulse, usually full and strong, is increased in frequency, but not in proportion to the number of respirations. There is a well-marked flush on the cheeks, producing a dusky, heavy expression of countenance, the remains of which are often apparent some little time after complete recovery. *Herpes labialis* is almost a diagnostic sign, and is said to be most marked on the side of the mouth corresponding to the inflamed lung. The urine is high coloured, of high specific gravity, and contains an increased amount of urates, whilst the chlorides are diminished or lost. Jaundice may be present in the early stages. Delirium, especially at night, and subsultus are not uncommon in severe cases. The duration of the disease is from five to eight days; the termination being marked by a crisis which may take the form of delirium, profuse sweating, epistaxis, or diarrhœa, the temperature at the same time falling precipitately to 100° , or almost to normal; then on the two following days it falls to, or even sinks below, the normal level; and the patient is well, unless (i) there be some further complication or relapse, or (ii) the upper lobes be involved when the crisis is delayed and is more gradual. Physical signs, however, of the third stage linger for some days after the patient has apparently recovered. The crisis is usually attended

by a return of chlorides to the urine. It should be remembered that death not infrequently occurs during the critical state, the patient passing into a 'typhoid' state before the end comes.

Physical Signs.—In the *first* stage there is some dulness, but it is not a marked feature. There is imperfect chest expansion ; the breathing is coarser than normal, and is attended during inspiration by a fine crepitation, which has been likened to the noise produced by rubbing one's hair between the fingers. This sign may, at times, be heard only at the end of a deep inspiratory effort, but it is pathognomonic when detected.

The *second* stage is characterised by signs which attend consolidation. Little or no air enters the air-cells, consequently crepitation ceases ; there is a dull percussion note ; and the vesicular murmur is changed to harsh, bronchial, or tubular breathing, whilst the vocal phenomena are exaggerated, or replaced by bronchophony and increased fremitus. In cases, however, where the main bronchial tube leading to the inflamed area is obstructed there may be an entire absence of the above signs.

The signs of the *third* resemble those of the second stage until such time as liquefaction of the inflammatory products occurs. Then the tubular respiration is changed to large moist sounds (subcrepitant râles and bronchial gurglings), which gradually disappear as the disease clears up. A somewhat dull percussion note persists for some little time after convalescence, and the aspect of the patient is not infrequently permanently dusky.

It is well to bear in mind that extensive pneumonia may exist in the interior of the lung, or at its diaphragmatic aspect, and yet none of the above physical signs be detected.

Complications.—**RESPIRATORY SYSTEM.**—Bronchitis and pleurisy. The inflammatory condition may extend to the larger bronchial tubes or to the pleura, with the characteristic signs of inflammation of these parts. Small irregular pneumonic abscesses may form. In debilitated subjects the disease may run a chronic 'smouldering' course for some

weeks, with the supervention of gangrene in small or large patches.

NERVOUS SYSTEM.—Convulsions in the initial stage are not uncommon in children ; whilst in adults a wild delirium is occasionally observed, often from the very onset of the disease, especially if the apices and upper lobes be involved.

Diagnosis.—1. FROM TYPHUS FEVER.—By the absence of petechial spots ; the face and conjunctivæ are not so dusky and congested ; and there is an absence of characteristic odour. 2. FROM TYPHOID FEVER.—By the absence of rose spots, and of tympanites, distension, diarrhœa, and other abdominal signs. 3. FROM ACUTE TUBERCULOSIS.—At times the diagnosis is very difficult. In pneumonia there should be a crisis between the sixth and eighth day. Stethoscopic evidence is not always reliable. We are mainly then guided by the history of the patient, his appearance, and the hectic character of the fever.

Prognosis.—Favourable, as a rule, in healthy, well-nourished subjects : hence the statistics of private practice are more favourable than those of hospitals. Still it is a disease which is attended by great mortality in elderly or intemperate patients, or those with debilitated constitution or physique, or where extensive tracts of pulmonary tissue are affected. The mortality is apparently as high in cases of unilateral as in bilateral pneumonia.¹ Cases of apical pneumonia are the most severe.

An unfavourable termination may be expected in cases in which there is dark, prune-juice-coloured expectoration ; in hyperpyrexia (105° or 106°) ; or in those patients presenting marked nervous symptoms, such as severe delirium, prostration, or carphology. Death is usually due to asphyxia or to syncope.

Treatment.—Rest in bed is absolutely necessary. No drugs have any effect in shortening the course of the disease. A purge (calomel gr. iij.) may be given at the onset. A robust patient with a full pulse will often derive much benefit from free bleeding in the early stage (leeches or venesection). Then treat symptoms as they arise. Antimony in small

¹ See Hadden, Mackenzie, and W. W. Ord : St. Thomas's Hospital Reports, 1891.

frequent doses has been recommended with a view to reducing fever¹ (Walshe). Alkalies with the iodine salts may be given to render the expectoration less tenacious, whilst ammonia and ipecacuanha are often useful from their expectorant action. Relieve the cough with hyoscyamus, tolu. Opium in small doses may at times be given with advantage, but not if there be much venous congestion, or a tendency to delirium. Dr. Ord, in view of the likeness of pneumonia to erysipelas, prescribes large doses of perchloride of iron during the acute stages. We have certainly seen benefit accrue to the patient from this treatment. Stimulants (ammonia or alcohol) are, as a rule, required at one or other period of the illness, and should then be freely given. Recently the inhalation of oxygen has been tried, with marked success, by Dr. Brunton and others. (See 'British Medical Journal,' 1892.)

As regards external applications, two opposite modes of treatment have their advocates. Some prescribe cold compresses or ice-bags to the affected lung, but we should hesitate to adopt this plan. Others apply blisters, or other counter-irritants, hot fomentations, and poultices. The latter have disadvantages from their weight and odour. The best results probably ensue from the application of an oiled lint jacket, with an outer padding of cotton-wool. Delirium should be treated by large doses of bromide of ammonium, or, if the pulse be good, by hydrate of chloral. Maintain the patient's strength by a generous and easily digested diet of milk, eggs, and good beef extracts. But it is essential that the stomach be not overloaded. Avoid the dangers of a relapse during convalescence by warm clothing, and, if possible, removal to some sheltered locality.

The treatment of such a complication as gangrene of the lung demands the free use of stimulants, nutritious food, and the inhalation of medicated sprays or vapours. Excision of the gangrenous portion has been advocated.

¹ R. Vin: Antim: Tart: $\mathfrak{m}\mathfrak{v}$.; Potass: Bicarb: gr. x.; Ammon: Carb: gr. iv.; Aquæ Camphoræ ad $\mathfrak{z}\mathfrak{j}$. Miscæ.

(b) **Catarrhal Pneumonia** (BRONCHO-PNEUMONIA ;
LOBULAR PNEUMONIA)

Definition.—An inflammatory affection of the lobules of the lungs, occurring mostly in scattered patches, and generally as a complication or extension of previous bronchitis.

Causes and Pathology.—Common in childhood, when it is generally acute, and follows an attack of bronchitis. Or it may be a complication of measles, rickets, or whooping cough. The bronchitis being the starting point, there may be secondary infection of the lobules by the inhalation of the products of inflammation from the larger tubes ; or the lobules may become involved by direct inflammatory extension to the alveoli, owing to the excessive irritability of their epithelial lining.

The affected foci first pass through a stage of hyperæmia, hence the lung appears studded with patches varying in size from a hemp seed to a filbert ; these patches being sharply defined, and giving the lung a mottled appearance on section.

The second stage is characterised by the blocking of the alveoli with the proliferated cells lining them, there being little or no fibrillated fibrin, the cell proliferation being the essential feature. In the third stage the invading elements undergo fatty changes, become liquefied, and finally absorbed ; or they undergo caseous degeneration, and so set up a form of chronic pneumonia, with breaking down of lung tissue. Subsequently true tubercular phthisis may supervene, owing to the invasion of the tubercle bacillus over the affected area.

Symptoms.—The disease being secondary to bronchitis, there have been previously observed all the signs and symptoms common to that condition. Then a sudden exacerbation of symptoms ensues, marked by rigor, increased dyspnea, flushed countenance, and painful cough. The temperature is high, as in croupous pneumonia, but markedly remittent. Expectoration, besides being difficult to obtain from children, is always scanty. It may, however, present the purulent characters of bronchitis, together with a slightly blood-tinged or rusty tint,

indicating extension of disease to the pulmonary alveoli. Recovery is gradual, and there is no critical day of the fever.

Physical Signs.—Not well-marked, apart from the bronchitis, unless the inflamed patches are numerous, close together, or have coalesced. If the patches be of large area, then there may be dulness on percussion; otherwise there is little or no impairment of percussion note. Careful auscultation, however, reveals the general large, moist sounds of bronchitis, with isolated and sharply defined patches, over which the respiratory murmur is harsh and tubular in character. If, however, the bronchi leading to inflamed infundibula be obstructed, no respiratory murmur at all is heard.

Diagnosis.—FROM CAPILLARY BRONCHITIS.—This disease, although attended by fever, lividity, and dyspnoea, presents most physical signs at the bases. The expectoration is purulent or muco-purulent, and there is an absence of tubular breathing.

Prognosis.—Always a grave disease, inasmuch as it supervenes on an affection which has already tried the patient's strength. The mortality in infants and very young children is heavy. A guarded prognosis should always be given in cases of constitutional weakness or disease (rickets, &c.).

Treatment.—The patient should be placed in a warm room, as in treatment of lobar pneumonia. A steam tent is always advisable. Blood-letting or other depletory measures are not to be commended. Apply warmth to the chest (cotton-wool, hot fomentations, &c.). The oiled jacket is an admirable substitute for the poultice, and is always well tolerated. The disease is essentially one which requires stimulants. Give the carbonate of ammonia, with ipecacuanha in small repeated doses.¹ Watch the pulse, and give brandy or other alcoholic stimulant in warm milk every two hours if the strength shows signs of failure. An emetic of vin. ipecac. (ʒss. to ʒj.) is often of great service in dislodging accumulated secretions of the bronchi, and is not so depressing as antimonial preparations. The diet should consist of warm, easily digested food,

¹ R. Ammon: Carb: gr. j.; Vin: Ipecac: mx.; Decoct: Cinchon: ad ʒj. Misce.

such as peptonised milk, eggs, and well-prepared beef extracts, or chicken broth.

CHRONIC INTERSTITIAL PNEUMONIA (FIBROID PHTHISIS)

Definition.—A chronic inflammation producing an increase of the connective tissue, subsequently developing into fibro-nucleated growth, which invades the peribronchial spaces, interlobular tracts, and causes obliteration of the air-cells and general contraction of the lungs.

Causation.—Rarely a primary affection, being usually secondary to pneumonia, chronic pleurisy, collapsed portion of lung, infarcts, or diabetes.

Pathology.—The first stage is characterised by hyperæmia and swelling of the ordinary connective tissue of the lung and its bronchial tubes (red induration) ; this is followed by hyperplasia of fibro-nucleated growth, which involves the peribronchial, interlobular, and intercellular areas. The inflammatory process generally extends to, in some cases from, the pleura, so that the lung is firmly attached to the chest walls. On section the organ is seen to be mapped out into islets of varying size, which are surrounded by bands of tough, fibrous tissue in which blood pigment is usually embedded (brown induration). Thus the lung is increased in actual bulk, though diminished in actual air capacity. Eventually the lung is converted into a fibrous mass, with scattered loculi representing the remnants of the lobular structures, and is not unlike the cut section of advanced cystic disease of the kidney. The disease is frequently confined to one lung.

The secondary effects of this condition are : (1) dilatation of larger bronchial tubes (*bronchiectasis*), due probably to retraction of the new fibroid tissue ; (2) a globular condition of the terminal tubes, due possibly to obstruction, with accumulation, and retention of secretions behind the obstruction ; (3) emphysema surrounding an indurated area ; (4) the formation of true cavities, as a result of breaking down of the adventitious tissue ; (5) enlargement and pigmentation of bronchial

lymph glands, and general pigmentation of the whole organ (black induration).

Symptoms.—Obscure in the early stages. If, however, after a history of recent pneumonia or pleurisy there is constant dragging pain, with retraction of the chest wall, over the site of the affection, together with dyspnœa, cough, and much expectoration of yellow or brownish offensive sputa, we may suspect that induration is taking place. Besides these symptoms the cough is often paroxysmal, and is aggravated by sudden change in position ; there are increasing anæmia and night sweats ; but there is no marked change in the pulse, nor in the temperature, until the disease has been long advanced, when the febrility assumes a hectic type ; hæmoptysis is frequent ; hypertrophy, and dilatation of heart cavities, lividity and bulbousness of the finger ends, and general dropsy occur in the last stages.

Physical Signs will much depend on the stage of the disease. There is usually some flattening and loss of movement of chest wall. Percussion is dull, and if there be marked dilatation of tubes the ‘*bruit de pot fêlé*’ is obtained. The breathing is feeble and distant, except over dilated tubes, where it may be cavernous. Similarly, moist sounds, varying in quality from râles to gurglings, are frequently heard.

Diagnosis.—The disease may be confounded with (1) chronic pleurisy ; (2) chronic tubercular phthisis. In all, the physical signs are much the same : the presence of signs of dilated tubes would almost preclude pleurisy ; whilst the history of the case—the apices being the earliest site of affection—and the presence of the tubercle bacillus, would be in favour of phthisis. It must be remembered, however, that in the later stages of cirrhosis, tubercular phthisis may be grafted on the original disease. Fibroid phthisis is, however, almost always unilateral.

Treatment.—Palliative mainly. Treat the case as one of tubercular phthisis. The patient should give up all injurious occupation. The strength should be maintained by stimulants and nourishing diet. Allay the cough, and promote expectoration. Cod-liver oil and the inhalation of turpentine vapours have been found most useful.

CHRONIC PNEUMONIC PHTHISIS

Definition.—A chronic disease of the lungs which, commencing in pneumonia, has tubercular disease engrafted on it, or *vice versa*.

Pathology.—It is difficult to reconcile the various conflicting views and statements of eminent pathologists and clinical observers. It is undoubtedly true that in cases dying of pneumonic phthisis there are evidences of catarrhal pneumonia, and careful staining of sections of lung will reveal giant cells, bacillus tuberculosis, and other evidences of tuberculous phthisis. There is also caseous degeneration of glands and neighbouring tissues.

The tree, however, 'is known by its fruit ;' and to us it is difficult to accept the hypothesis that tubercle in one case produces the catarrhal signs and symptoms of ordinary phthisis, and in another the chronic pneumonic consolidations of the disease in question. It seems to us much more probable, in accordance with the characteristics of disease, that it is not a true phthisis in the usual acceptation of the term ; but either (1) a pneumonia occurring in a subject predisposed to tubercle, and in this case producing a rapid destruction of tissue, as erysipelas may do in a debilitated subject ; or (2) a primary pneumonia, which, from constitutional defects, is slow of resolution, and is complicated by the advent of the tubercular process.

Symptoms.—Vary, according to the character and extent of the lesion, resembling in part those of tubercular phthisis, chronic pneumonia, or bronchitis.

Treatment.—The same rules should guide us as are described under Pneumonia.

GANGRENE OF THE LUNG

Definition.—Death *en masse* of a certain portion of the lung tissue.

Causation.—The condition may be : (a) secondary to some inflammatory disease of intense character, such as pneumonia, specific fevers, when it is probably akin to noma. In such cases there are other predisposing factors, *e.g.* chronic alcoholism,

diabetes, senility, syphilitic disease of the blood-vessels. (b) It may result mechanically from pressure of an aneurysm, new growth, hydatid cyst, the presence of food or other foreign bodies in the bronchi, or from hæmorrhagic infarct. Or (c) it may have a septic origin from diphtheria, or malignant new growth of mouth, fauces, and upper air-passages; from extension of local abscess; it may therefore occur as a complication in pyæmia or the puerperal state.

Pathology.—Two forms recognisable: (a) diffuse; (b) circumscribed. In the former, the disease may attack large tracts of lung tissue, or even involve the whole of one lobe. There is no limiting line, the necrosed lung tissue merging at its circumference into pneumonic and congested areae. The gangrenous tissue on section is seen to be disorganised into a soft, juicy, greenish-coloured mass with a horribly offensive discharge. In the circumscribed form, the periphery of the lower lobe of the lung is the most usual site. The diseased tissue is in patches, with distinct limiting zones of consolidation. The patches may break down, discharging their contents by neighbouring bronchi; or they may coalesce with similar neighbouring patches, forming varying sized excavations which occasionally open into the pleural cavity.

Symptoms.—Unmistakable when a gangrenous patch opens into and discharges itself by a bronchial tube; the breath and expectoration being horribly offensive, resembling decomposed animal matter. The sputa are equally fœtid, of a dirty brown or blackish tinge, and contain histological elements of lung tissue.

There is great prostration and dyspnoea, with a small and feeble pulse, and a tendency to the 'typhoid' state.

The **physical signs** are those found in consolidation, with subsequent breaking down of lung tissue and the formation of cavities.

Diagnosis.—The condition may be confounded with *Bronchiectasis* and with *Pyo-pneumothorax*. In the former disease there would be a history of previous chronic bronchitis; and in the latter we must depend on the history and the physical signs.

Prognosis.—Generally unfavourable, especially in the diffuse variety; death generally ensues from exhaustion, hæmorrhage, or extension to the pleural cavity.

Treatment.—Maintain the strength by stimulants and beef-tea. Prescribe antiseptic, odorous inhalations—*e.g.* carbolic acid, creasote, turpentine. The latter may be given internally both as an antiseptic and stimulant (Begbie). Surgical excision of a gangrenous patch near the surface has been successfully practised.

PULMONARY TUBERCULAR PHTHISIS (CONSUMPTION)

Definition.—A destructive inflammatory disease of the lungs, usually of a chronic nature, commencing in the connective tissue of the alveoli, or in the peribronchial lymphoid tissue, and due to the presence of granular neoplasms called ‘tubercles.’

Causation.—(a) **PREDISPOSING.**—*Age.*—Most frequent between fifteen and thirty-five; no period of life, however, is exempt; and though comparatively rare after fifty, cases are recorded as occurring in patients between the sixtieth and seventieth year, when it is found not unusual for several members of the same family to succumb from tubercular phthisis during the same decade. *Sex* has probably no preponderating influence, except that females are more liable when the disease is hereditary; whilst males, from their greater exposure, are more prone to the acquired disease. *Climate.*—A cold, moist climate is most favourable to the spread of the disease, as it is comparatively unknown in the torrid and in the arctic zones. *Heredity.*—A most powerful predisposing factor, not only as regards direct transmission of tubercular disease from parent to child, but also by the descent of syphilis, scrofula, and other wasting diseases, rendering the offspring more prone to acquire tuberculosis. *Wasting Diseases*, such as diabetes, Addison’s disease, exhausting fevers, anæmia, &c. *Diseases of the Respiratory Tract*, such as bronchitis, pneumonia, pleurisy, to which must be added measles, whooping cough, and injuries to the lungs.

Certain Trades and Occupations, notably compositors, milliners, tailors, millers, workers in textile fabrics, and others. *Unhealthy Hygienic Surroundings*, such as impure atmosphere, deficient ventilation, and undrained, damp soils.

(b) **EXCITING.**—The tubercle bacillus of Koch (see Tubercle, p. 43). This conclusion is based on the following data—viz. the bacilli are found in the sputa, in the tubercles themselves, and in the various tissues and organs of the body of patients suffering from tubercular disease, and especially are they ‘found where the tubercular process is commencing or spreading’ (Flügge); after being stained by fuchsin and other reagents the bacilli retain their stain, and are thus distinguished from putrefactive organisms; they have been isolated and separately cultivated; they flourish at the temperature of the human body and in animal media only. If introduced into the human body, either by inhalation in the form of a spray, or by inoculation, sooner or later the characteristic lesions of tuberculosis are found. Artificial cultivations of the bacillus through numerous generations are equally as infective as the first.

Pathology.—Any part of the lung may be primarily affected; the apices are, however, most frequently the first attacked, especially the left, owing to stagnation of air as a result of weak respiratory movements.

Infection usually commences in the finer bronchi, and extends (1) to the alveoli; or (2) to the peribronchial connective tissue.

(1) If it extend to the alveoli, the diseased part is studded with solid masses of lung tissue, which have a tendency to become grey or opaque in the centre and to coalesce with neighbouring masses at the periphery, the whole apex thus becoming condensed and solid. Microscopically the small tubes leading to the alveoli are plugged with inflammatory exudation, whilst the alveoli themselves are filled, as in pneumonia, with a mass which consists partly of the alveolar epithelium, and partly of migratory leucocytes and red blood-cells. Definite tubercles, with typical giant cells, may be found in the bronchial or in the alveolar walls. In a subse-

quent process the structures involved undergo molecular death, the cells becoming granular, homogeneous, and cheesy (caseous necrosis).

Subsequently the caseous material softens, and exudes a thick, muddy fluid, often containing elastic tissue and other histological elements of the affected air-passage, the fluid ultimately discharging itself into a neighbouring bronchus, and is expectorated. The cavities thus formed vary in size according to the extent of tissue involved, or according as the original cavities remain isolated or coalesce with their neighbours. A fresh cavity has irregular walls lined with recent or caseating tubercle, with, at times, blood-vessels extending from one side to another. Subsequently the walls become smoother, more defined, and lined with a pyogenic membrane. The destructive process described above may be arrested at the caseous stage, when the inflammatory products are partly absorbed, and the remnants become infiltrated with lime salts; or arrest may occur at the stage of vomica, when the contents having been discharged, the walls contract and become limited by indurated tissue.

(2) In this form the initial lesion commences also in a plugged bronchiole, and extends to the peribronchial connective tissue by means of the lymphatics. This tissue becomes hypertrophied, forming bands or areas which surround crepitant lung tissue; subsequently the inflammatory process extends to the surface, causing thickening of the pleura and firm adhesions at the apex. Tubercles are found scattered throughout this inflammatory connective tissue and round the bronchi.

Other tubercular deposits are generally found in the peritoneum, cerebral meninges, liver, kidneys, and intestines. In the bowel they often give rise to ulcerations, which for the most part have their long axes transverse to the gut. (See Coats's 'Manual of Pathology.')

Symptoms.—Three stages are ordinarily recognised. The **FIRST STAGE** (incipient consolidation) is generally ushered in by dyspepsia, slight cough, and some fever. The cough is not at first severe, but is usually attended by slight mucous ex-

pectoration ; there may be a little dyspnœa ; the pulse becomes quickened, especially with exertion ; and there is a slight amount of fever, more marked towards the evening (bacillary infection). These conditions may continue for months, but increased cough and slight hæmoptysis mark a progressive aggravation of the disease. The cough then becomes more troublesome, the expectoration is muco-purulent, and night sweats supervene. The above symptoms are usually observed in the first stage of a gradually developing case ; but it not unfrequently happens that the disease has bronchitis, pneumonia, or some exhaustive fever as a precursor, or that sudden hæmoptysis may be the first symptom.

The SECOND STAGE is marked by an aggravation of all the previous symptoms. Hectic is now pronounced, the cough more troublesome, whilst the expectoration becomes markedly purulent, being often expelled in well-defined, creamy, nummular masses. There is often a well-defined flush on the cheeks, and marked brilliancy of the eyes. The skin, which is hot and pungent during the day, is sodden and bathed in sweat during the night, and the finger ends become bulbous, with curved nails. Pain in a localised area at the apex is often complained of.

The THIRD STAGE is characterised by increase of cough, of night sweats, of emaciation, and of expectoration. Severe attacks of alarming hæmorrhage are common ; pain at the summit of the lung is more pronounced ; diarrhœa frequently sets in ; whilst nausea and vomiting, probably as the result mainly of the cough, are also met with. During this stage, yellow elastic tissue is frequently found microscopically in the sputa.

Death may result in this stage from : (1) *perforation of the pleura* (pneumo-thorax) ; (2) *hæmorrhage* (rare) ; (3) *severe diarrhœa*, or even perforation of the small intestine from tubercular ulceration ; (4) *severe and progressive emaciation* ; (5) *sloughs or bedsores*. Usually the mental condition remains clear to the last.

Whatever the stage may be, there are four symptoms which suggest phthisis without examining for physical signs,

viz. : persistent cough, emaciation, night sweats, and hæmoptysis.

Physical Signs.—**FIRST STAGE.**—*Consolidation.*—At the affected apex there is loss of expansion, dulness on percussion, and increased vocal phenomena. The auscultatory signs vary. The breath sounds may be diminished over one area, and increased over another ; or the breathing may be interrupted, ('eog-wheeled') or tubal ; whilst in neighbouring unaffected regions it is puerile, owing to increased action of healthy surrounding tissue. A prolonged expiratory murmur, together with a coarse character of breathing, both with inspiration and expiration, are, however, usually present. Rhonchi may be heard especially after coughing ; and not infrequently small cracklings and ereakings, as a result of local pleurisy, are detected. It should be remembered that all the above physical signs may vary in intensity, according to the extent of the lesion or to the amount of neighbouring emphysema.

SECOND STAGE.—*Softening.*—The supra- and infra-clavicular flattening becomes marked ; dulness is more extended and more pronounced ; bronchial breathing more intense, and accompanied by sharp cracklings (suberepitant râles), which remain after, and are unaffected by, coughing. Owing to the retraction of lung tissue at the diseased apex, the preeordial dulness is increased, and even the heart itself may be displaced.

THIRD STAGE.—*Cavity, or Vomica.*—The signs are those usually produced by cavities of various sizes. Slight percussion yields the *bruit de pot fêlé* (cracked-pot sound), provided the cavity be near the surface ; if deeper, a heavier percussio yields a somewhat dull note. Various conditions, however, modify this physical sign. For its full development it is requisite that the cavity be large and near the surface ; that it communicate freely with a patent bronchus ; that it be surrounded by consolidated lung tissue ; and that it contain air and a little fluid. The auscultatory signs vary also according to the size of the cavity. If it be small, then the breathing is cavernous, resembling somewhat that which is

heard over a dilated tube ; if it be large, and moderately full of thin pus, then gurgling is a frequent sign. If the excavation be of very large size the gurgling is more pronounced, and 'metallic tinkling' is often present during the act of coughing or speaking. The characteristic vocal sounds are pectoriloquy or cavernous whisper. All these signs may be modified, or even absent, owing to varied and changing conditions of the excavated lung. In a well-advanced case three physical signs, however, are almost invariably present, viz. : (1) apical dulness, with (2) large moist sounds ; and (3) pectoriloquy.

Diagnosis.—Hæmoptysis may occur in many diseases or conditions other than phthisis. But occurring in a patient with cough, dyspnœa, progressive emaciation, purulent sputa, night sweats, and intermittent fever, the diagnosis is tolerably clear. It would be confirmed by finding signs of softening at one or both apices, and by the detection of the pathognomonic bacilli in the sputa.

Prognosis.—Mostly unfavourable. Recovery may take place in the first or early part of the second stages. The prognosis is rendered more grave by : (1) hereditary predisposition ; (2) unhealthy occupations or surroundings ; (3) complications, such as laryngeal infection, capillary bronchitis, pneumo-thorax, profuse hæmoptysis, amyloid affection of kidneys and other organs, diarrhœa.

Complications.—The complications are numerous and varied. Some of them are due to tubercular invasion of organs and tissues other than the lungs ; some are due to accidents common to the progress of the disease ; whilst, again, others are part of the febrile process.

No classification of complications can be entirely accurate and satisfactory, owing to the variety of causes from which they originate. And it will be seen that in many instances the complications themselves are described as separate diseases in various parts of this work. The following enumeration is, however, put forward on an anatomical basis as being most convenient.

(a) **RESPIRATORY TRACT.**—(1) Tubercular deposit in the mucous membrane of the *larynx* and *trachea*, leading to

ulcerations and necrosis of cartilages, and occasionally severe hæmorrhage. Tubercular infiltration may first originate, though rarely, in the larger air-passages ; and in that case the course of the disease is generally a rapid one. Usually, however, it is secondary to the lung affection. (2) *Pneumo-thorax*, from rapid extension of ulceration from a superficial cavity. This occurs most frequently in very acute cases, before a local adhesive pleuritis has had time to arrest the process of excavation. The gravity of this complication is increased if it occurs in the less diseased side. It is usually followed by *pyo-pneumothorax*. (3) *Pleurisy* itself may be due to the deposit of tubercle, and often gives rise to distressing pain and dyspnœa. Frequently, however, as shown above, it is rather a beneficial process, preventing a fistulous communication between a tubercular excavation and the pleural cavity. (4) *Profuse hæmorrhage*, from a ruptured aneurysmal dilatation of a branch of the pulmonary artery. Occasionally the hæmorrhage may burst through into the pleural sac, or extravasate into the lung tissue. It is usually, however, coughed up, often in large quantities. It is noteworthy that death from the immediate effects of pulmonary hæmorrhage is rare, although by the exhaustion it causes it undoubtedly hastens the fatal termination of phthisis.

(b) CIRCULATORY SYSTEM.—(1) *Cardiac atrophy* may occur as part of the wasting process. (2) *Valvular incompetence* is rare ; but it may result from adhesion of the heart walls to neighbouring lung, or from dilatation of the heart's cavities secondary to the shrinking of a large pulmonary excavation. (3) *Thrombosis* of various trunk veins, notably the femorals. (4) *Amyloid degeneration* of various organs.

(c) DIGESTIVE SYSTEM.—(1) *Aphthous ulcerations* may occur in the buccal cavities. (2) *Vomiting* is frequent : in the early stages it is caused by the paroxysms of coughing ; in the later stages it is due to catarrh of the stomach. (3) *Diarrhœa* may be present, either as part of the general febrility, or due to (4) *Tubercular ulceration* of the small intestines. (5) *Hæmorrhages* from the bowel may take place from time to time, as a consequence of ulceration ; but is somewhat rare. (6) *Peritonitis*

is apt to occur, either from perforation of an ulcer, or from tubercular invasion of the peritoneum itself. (7) *Fistula in ano* is a somewhat more frequent complication. It is caused by tubercular ulceration extending from the rectum into the ischio-rectal fossa ; it is often, however, an occurrence antecedent to phthisis. (8) *Fatty infiltration of the liver, ascites, and jaundice* are occasionally met with in advanced chronic cases.

(d) GENITO-URINARY SYSTEM.—(1) *Tubercular infiltration of the kidney* is not uncommon. In acute phthisis the tubercles are found in the grey miliary stage ; in a chronic case they undergo caseous changes, and form irregular abscesses containing cheesy matter and pus (*scrofulous kidney*, see p. 551). Similar deposits may be found in the urinary bladder, causing (2) *albuminuria* and (3) *hæmaturia*. Albuminuria, however, is not necessarily symptomatic of tubercular disease of the kidney ; it may supervene as a symptom of the general febrile condition. (4) *Tubercular epididymitis* is an occasional complication. In most of the cases which we have seen it would appear to have been the primary lesion.

(e) CUTANEOUS SYSTEM.—(1) *Œdema* of various parts, and (2) *Bedsore*s are common in the advanced stages of chronic cases. (3) *Sweats*, especially at night, may be so severe as to rank as a complication.

(f) NERVOUS SYSTEM.—(1) *Tubercular meningitis* and (2) *Delirium* ; the latter being due to fever.

(g) GENERAL TUBERCULOSIS.—In the course of an ordinary case of chronic pneumonic phthisis, the disease may suddenly become acute, and present symptoms of invasion of bones, joints, and various organs and tissues of the body which had previously appeared exempt from invasion.

Treatment.—HEALTHY SURROUNDINGS.—The patient should be removed from harmful occupation. His rooms should be kept at an equable yet warm temperature, with an abundance of fresh air ; this applies with especial force to the bedroom. Gentle exercise on foot should be recommended ; he should guard against cold winds, or any sudden change of temperature. As regards the selection of climate or health resort, much

difficulty presents itself. Residence in high altitudes has lately been much extolled. The localities most frequently advocated are Davos, St. Moritz, the Scotch Highlands, the Rocky Mountains, Colorado, Quito, and the Andes. The remedial properties of the atmosphere at these elevated stations depend on its dryness, stillness, great purity, and lightness. Many patients, however, return with little or no benefit. Each case should be carefully considered on its own merits. Cases in which the circulatory system is weak, or in which there is a tendency to hæmorrhage, are not suitable. We have seen perhaps the best results from prolonged voyages on the ocean. Other patients do best at relaxing and sedative health resorts, such as Torquay, Cornwall, Pau, Madeira ; but as a rule these climates are depressing, enervating, and tend to favour hæmorrhage. Again, a stimulating climate, where the air is dry and bracing, may be desired, such as Margate, Folkestone, Isle of Wight, Mentone, Egypt, New Zealand. Complete change of home and climate should be undertaken in the early stages only. It is worse than useless to send an invalid away in the last stage. (See Douglas Powell : ' Diseases of Lungs.')

DIETETIC.—The dietetic principles to be kept in view are, that the food should be simple, nourishing, easily digested, and non-stimulating. Thus milk, eggs, farinaceous food, fish, and fowl are all admissible.

MEDICINAL.—As regards drugs, cod-liver oil is of great advantage in the early stages, most probably, however, as a food. It increases the bodily weight, improves the appetite, and causes diminution in cough and sweat. It should be discontinued, however, in the case of those patients in whom it causes nausea or loss of appetite. Begin with small doses (5j.), and gradually increase them. Its disagreeable taste may be masked by mixing it with equal parts of honey and lime juice, or it can be mashed up with potatoes. If it cannot be taken by the mouth, inunction may be tried. *Dyspepsia*, so common at all periods, may be met by vegetable tonics with alkalies. Iron is indicated if there be much anæmia, the ferri et ammoniæ citras (gr. v.) being perhaps most easily tolerated. Arsenic, the hypophosphites of soda and ammonia are also of

great value. Our further aim should be directed to relieve urgent symptoms. Treat the *Cough* by morphia, opium,¹ chlorodyne, ipecacuanha. Lozenges are probably the most convenient form. Be careful not to derange the digestive system by these drugs. For *Night Sweats* nothing seems so efficacious as a cool, fresh-aired bedroom, with frequent changes of body and bed linen. The body may be sponged with tepid water, to which dilute sulphuric or acetic acid can be added. We are not convinced of the great advantage of belladonna by the mouth, although atropine injected hypodermically is often of distinct service. *Vomiting* in the early stages is often the result of paroxysmal attacks of coughing; in the later stages it is caused by catarrh of the stomach, and therefore generally comes on after food. It is best met by alkalies, especially the bicarbonate of soda, in conjunction with dilute hydrocyanic acid. *Diarrhœa* similarly is seen in the early stage, when it frequently depends on irritation of partially digested food. It is relieved by a change of food, and a slight laxative (pulv. glycyrrhizæ co., or pulv. rhei co.); but in the later stages, when accompanied by a raw, glazed tongue, it is significant of ulceration of the bowels, and is controlled by sulphate of copper, or by bismuth, hæmatoxylum, pulv. kino comp. *Laryngeal ulceration* is best treated with morphia insufflation, sprays of solution of hydrochlorate of cocain (five per cent.), or the direct application of a solution of nitrate of silver (gr. xv. to ʒj.).

The skin over the sacrum, ilia, and shoulders must be kept free from bedsores by evaporating lotions, air cushions, &c.

In all stages it is of the utmost importance that suitable receptacles filled with disinfecting fluid be provided for the sputa.

We are not yet in a position to pronounce definitely as to the value of Koch's treatment of injecting *tuberculin*. We are of opinion, however, that if the method be tried, cases should be selected which are in the early stage, and as free

¹ R. Liq: Opii Sed: mxxv.; Acid: Hydrocyanici Dil: mlij.; Succ: Limonis ʒij.; Syrup: Tolu: ʒss.; Syrup: Rthæados ad ʒj. Misce; ft. Linctus. ʒj. for a dose.

from complications as possible. We have certainly, in a limited experience, seen favourable results in two cases.

ACUTE MILIARY TUBERCULOSIS

Definition.—A general tubercular infection of the whole of the organs and tissues of the body, especially pronounced in the lungs, and running an acute and rapidly fatal course.

It is perhaps not correct to place this disease in the category of Diseases of the Respiratory Organs, but as the lungs are invariably involved, and to a greater extent than any other organ, it is convenient to consider the condition here.

Pathology.—The lungs are found studded throughout, both on the pleural surfaces and in their interiors, by miliary tubercles, grey and translucent in appearance, and all in the same condition of growth and appearance. The bronchial, mesenteric, and other lymphatic glands are enlarged, and possibly caseating. Around each tubercular deposit the tissues are congested and œdematous. Similar deposits are found in the pleuræ, peritoneum, pia mater, joints, intestines, and in the liver, kidneys, and other glands. Death usually supervenes before any degenerative or destructive changes can occur, but occasionally a yellow focus of degeneration (fatty) may be detected in some of the tubercles.

Symptoms.—The disease commences with fever, prostration, and rigors ; then follow dyspnœa, with rapid breathing, increased pulse-rate, thirst, dry tongue, perspiration, with constipation or diarrhœa. In other words, the condition is such as one would find in acute septicæmia. Subsequently there may be some cough without expectoration, or hæmoptysis and lividity of face and extremities ; the temperature increases (to 104° or more), with distinct morning remissions ; sordes form on the teeth and lips ; and death is preceded by subsultus, delirium, and coma.

There is frequently in such cases a family predisposition to tubercular disease.

Physical Signs.—There may be absolutely *none*, so far as the lungs are concerned. Generally, however, the apices show

signs, sooner or later, of acutely advancing consolidation. Auscultation may yield no sign beyond that of slight bronchial catarrh. It is obvious, however, that if the patient survive until breaking down of lung tissue occurs, physical signs of this condition would present themselves.

Diagnosis.—FROM ENTERIC FEVER.—In both there is high fever of the remittent type, together with enlarged and tender spleen and distended belly. But in acute tuberculosis the tenderness is not more pronounced in the right iliac fossa, the stools are not pea-soupy, the breathing is more rapid, the temperature curve more even, and there are no spots.

FROM PNEUMONIA.—In tuberculosis, herpes and rusty sputa are seldom found. Consolidation of the lungs, if it be present, is usually at the apices; whilst the increased frequency of respiration is not accompanied with any marked dyspnoea at the onset. In pneumonia, again, one would expect a crisis about the sixth or eighth day.

Treatment.—The disease is invariably fatal. Little can be done beyond supporting the patient's strength by beef-tea, or by nutritive enemata, and the free use of stimulants.

MALIGNANT DISEASE (NEW GROWTH)

Any form of malignant new growth may invade the respiratory tract, and involve the bronchial tubes, air-cells, or pleura.

Causation.—*Age.*—Sarcomatous new growths may appear at any age. Carcinomata (medullary or scirrhus) affect adults of middle age only. *Sex.*—More common in males than females.

It is rarely a primary affection, but usually *secondary* to malignant disease commencing in the mediastina or in some distant organ, such as the mamma, stomach, or liver. Cancer is most frequently of the medullary type.

Symptoms and Physical Signs.—Variable, according to part of respiratory apparatus which is affected. Thus the signs may resemble those of an extensive pleuritic effusion, or pulmonary consolidation, if any large area be involved; or of

bronchial catarrh, with bronchial obstruction, when the growth involves any of the larger tubes. Look for signs of pressure on large venous trunks, or on nerves (recurrent laryngeal, intercostals, or sympathetic); these would be almost diagnostic in the absence of symptoms of aneurysm. Examine the root of neck and axilla for involved glands. Pain is not necessarily present; indeed, it is often absent. Profuse hæmorrhage may occur, should any of the larger vessels be eroded. Emaciation with typical cachexia are soon apparent. The temperature is normal, unless the disease is accompanied by secondary inflammatory processes.

Treatment.—Entirely palliative. Treat symptoms as they arise. Our aim should be directed to the relief of pain chiefly by opium, and to the administration of good, nourishing food. Surgical procedures are not advisable unless urgently demanded.

SYPHILITIC DISEASE OF LUNGS

Definition.—A secondary affection of the lungs due to syphilitic poison, producing symptoms resembling chronic, fibroid phthisis.

Pathology.—The disease shows itself: (1) as gummatous tumours; (2) as non-vascular, caseous nodules somewhat resembling tuberculous growths; or (3) as tracts of cicatricial tissue, involving especially the peribronchial spaces and fibrous septa.

Symptoms are obscure; none are diagnostic. Persistent cough with expectoration and emaciation are usually present. Large hæmorrhages occasionally occur, owing to extensive and rapid ulcerations. Look for evidence of syphilis in skin, bones, iris, and elsewhere. The disease is very rare, especially when we remember the prevalence of primary syphilis.

Treatment.—A long-continued course of iodide of potassium is necessary. Liq. hydrarg. perchlor. may be added, unless contra-indicated by anæmia, or other general condition. Cod-liver oil seems to be of especial value. The cough should be treated by usual remedies.

HYDATID

Primary hydatid of lung is very rare. The disease usually occurs as an extension from some other organ, notably the liver. The parasite, however, may develop in the lung, and is then generally in the form of a single cyst in the tissues surrounding bronchi or infundibula. The cyst may vary in size from that of a grape to a goose's egg, and is more frequently situated at the base of the lung.

Symptoms and Physical Signs.—Not marked until the cyst assumes large dimensions, or unless it is near the surface of lung. The symptoms produced are mainly those due to the pressure on surrounding tissues, and the high tension of the cyst. Thus there may be hæmoptysis, bronchitis, a circumscribed pneumonia, or pleurisy. When the parasite approaches the surface it pushes all structures before it, cartilages and bones even becoming bulged, and forms a rounded, sharply defined, tense tumour, which is free from pulsation, and occasionally a marked hydatid thrill can be elicited.

Subsequently the cyst may shrivel and dry up, owing to the death of the parasite; or it may rupture and discharge itself, before or after suppuration, through a bronchial channel, or into the pleural or abdominal cavity, or externally.

The detection of hooklets in the discharged contents is, of course, diagnostic.

Treatment.—Aspirate if feasible; but no exploratory puncture should be advised, unless (1) the diagnosis be almost unquestioned; (2) the sac be near the surface; and (3) pressure signs urgent.

Treat cough, pain, and pyrexia with usual remedies.

EMPHYSEMA

Definition.—A pathological condition, consisting of extreme dilatation of the air-sacs and attenuation of their walls, with or without rupture and extravasation of air into the interstitial connective tissue.

Causation.—The essential cause is the weakening, or even destruction, of the elastic tissue of the alveoli. Two distinct exciting causes bring this about, viz. : prolonged and forcible strain on the lungs ; and fatty changes in the minute tubules and infundibula. Thus it occurs as a sequel to whooping cough and chronic bronchitis ; it is seen in athletes, divers, glass-blowers, and others whose occupations entail prolonged strain on the lungs. It also is symptomatic of fatty and other degenerative changes of disease and advanced age. Occasionally the two factors of strain and fatty degeneration occur together in the same individual, as in chronic bronchitis ; and in the contraction of old excavations of the lungs, the alveoli surrounding these lesions becoming abnormally distended. This is regarded as a compensatory condition.

Pathology.—Two varieties are recognised : (a) Vesicular ; (b) Interlobular.

In *Vesicular* emphysema the lungs are expanded, distending the chest and hiding the heart ; they are soft, yet elastic, to the touch, and float in water like air cushions. Their appearance is generally pale and bloodless, with streaks of pigment along the more prominent septa. Microscopically the air-cells are found to be overdistended ; the walls separating individual cells are thinned and possibly ruptured, or in severe cases almost absent, so that neighbouring air-cells freely intercommunicate, or rather two or more alveoli are made into one. A further progress is marked by the fusion of, and communication between, two or more infundibula, thus forming somewhat smooth cavities, in which little or no evidence of air-cells remains beyond a slight irregularity of the internal surfaces, which are not infrequently pigmented. As a result of this overdistension the capillary plexus becomes stretched, narrowed, and in parts obliterated. The surfaces, also, on which this plexus should have ramified are thus actually diminished by destruction of the septa ; and finally the elastic elements of the air-vesicles are partially destroyed. In advanced cases the smaller bronchial tubes also share in the dilatation. These conditions, though probably mainly brought about by violent and prolonged expiratory efforts, are doubt-

less in great measure due to degenerative changes (fatty, gouty) in the alveolar walls, as already stated.

The *Interlobular* form is seen in children only, in whom the interlobular spaces and the tissues contained therein are well marked and plentiful. Rupture of the air-cells takes place after violent coughing or other expiratory strain ; the air which thus escapes extravasates into these interlobular and peribronchial connective tissues, and frequently finds its way to the root of the lungs, posterior mediastinum, and even upwards to the neck. At times the change is limited to a few infundibula and the adjacent branches of the bronchial tree.

Symptoms are mainly those of dyspnœa, cough, imperfect aëration of blood, and emaciation. The dyspnœa varies in severity. It may be paroxysmal, being accentuated by exertion, especially in high altitudes, by dyspepsia, or by bronchial catarrh. The face, usually pale, is often, after a bout of coughing, blue or even purple, whilst the superficial veins of the neck and head are engorged. Emaciation is progressive, being in no small measure due to disturbance of the digestive organs. The bowels are constipated. Beyond these symptoms it may be noted that the cough is attended by scanty frothy expectoration, and that there is no pyrexia unless the disease be accompanied by bronchitis, pneumonia, or other complications. As a result of the impeded circulation, passive congestion of the liver ('nutmeg liver') and of the kidneys (albuminuria) supervenes, although œdema of the lower extremities is less common than in mitral disease.

Physical Signs are important. The chest is large and barrel-shaped ; the sternum bowed ; the costal arch widened ; the liver and spleen are pushed down ; the heart's apical impulse is masked and overlapped ; whilst epigastric pulsation is usually well-marked. Further, the rise and fall of the chest walls during inspiration and expiration are restricted, whilst the extraordinary muscles of respiration are exaggerated in their action. On percussion the note is full and almost tympanitic, especially in the upper thoracic regions ; the heart, liver, and spleen areas of dulness are diminished, owing to the overlapping lungs. On auscultation the inspira-

tory murmur is short and enfeebled ; the expiratory murmur being prolonged and not infrequently attended by râles, or dry, 'cooing' rhonchi. The vocal resonance is diminished.

Diagnosis is easily made by the alteration in the size and shape of the chest ; the effacement or partial obliteration of cardiac and liver dulness ; the high-pitched percussion note, and the prolonged expiratory murmur.

Prognosis.—The disease is not immediately fatal. Patients even survive to a good age. Duration of life will depend on the presence or absence of bronchitis, or cardiac or renal diseases.

Treatment.—Mainly palliative. Avoid all conditions which may act as exciting causes to dyspnœa. Thus we should forbid severe exertion, and exposure to rough, cold winds. The clothing should be warm but not too heavy ; flannel is best. If possible, the patient should reside in a climate which suits him, the experience of the individual being the best guide. Diet is important : meals should be scanty, with short intervals ; large meals at long intervals being especially injurious. Tonics are serviceable, especially arsenic and iron ; whilst strychnia has been recommended with a view to give tone to the overstrained muscular fibres of the bronchi.¹ If emaciation be a prominent symptom, cod-liver oil is often of great value, especially when applied as an inunction over the chest.

Beyond this it is necessary to treat the asthma or bronchitis, of which emphysema is so often a sequel (see Bronchitis ; Asthma). Spasm may be met by antimony, ether, or lobelia. Chloral is also of great value, but should be given cautiously if there be much lividity or signs of a feeble heart.

COLLAPSE OF THE LUNG

Causation.—The causes of this condition may be classified into : (1) HEREDITARY OR CONGENITAL ; (2) EXTERNAL PRESSURE on the lung ; (3) INTERNAL OBSTRUCTION ; (4) EXHAUSTION.

¹ R. Liq: Strychniæ miiij. ; Tinct: Ferri Perchlor: mx. ; Spt: Chloroformi mxv. Aquam ad ʒj. Misce.

(1) HEREDITARY COLLAPSE (*Atelectasis*) may occur when for any reason the lungs are not sufficiently expanded at birth. It is seen in enfeebled or rickety children.

(2) EXTERNAL PRESSURE may occur on one or both lungs ; or it may take place over a more or less limited area of a lobe. Such pressure may be exerted by pleuritic effusion, pyo-thorax, hydrops pericardii, aneurysm, malignant or other new growth, and hydatid cyst. It may also be produced by tumours, or by collections of fluid originating in relation with the diaphragm, or the abdomen, or with any of its organs.

(3) OBSTRUCTION to the entrance of air to the lungs may arise from any disease which causes stenosis of the upper air-passages, of the nose, larynx, trachea, or bronchi. It is obvious that the higher up in the respiratory tract at which the obstruction exists, the more extensive the lung collapse, and the greater tendency to symmetry of this condition. Obstruction may, however, exist in the larger or smaller bronchi, and then the collapse of lung is limited to that lobe or lobule to which the impervious tube leads, and the lesions are therefore scattered. The actual cause of this condition may be some new growth originating inside the tube ; but most frequently it consists of a plug of tenacious mucus, which is drawn further in at every inspiration, the expiratory efforts being unable to dislodge it. Or, this mucous secretion may be so placed as to act as a valve opening outwards, by which the air from the infundibulum beyond can escape, but past which no oxygen is allowed ingress. The imprisoned air beyond this obstacle is of great tension at first, the air-sacs being inflated ; but after a time the air is absorbed by the surrounding tissues, and the lobule becomes collapsed.

(4) EXHAUSTION.—It is well known that patients dying from prolonged and exhausting fevers, such as typhoid, breathe in a very shallow manner for some time prior to the fatal event ; this being apparently due to both muscular and nervous exhaustion. Post-mortem the lungs in such cases often present the typical slaty colour and condition of collapse. Whatever the exact causation may be, we have also seen a similar collapse of lungs in patients dying from

typhoid fever and other febrile diseases, to whom the graduated bath had been administered as a method of reducing hyperpyrexia.

Pathology.—The collapsed portion of the lung is usually of a distinctive purple grey, or rather a slate tint. It is airless to the touch, and consequently does not crackle under the fingers, and feels generally limp and dry. Here and there the lung tissue shows signs of consolidation, and to all appearances is pneumonic; but this condition is not necessary; it merely shows that marked congestion had occurred in patches.

The extent of the collapse varies. In some cases, say after a prolonged pneumo-thorax, the lung is represented merely by a mushroom-shaped mass, no larger than an ordinary apple, fixed to the bronchus as by a footstalk. In others the lung lies flabby and flattened in the furrow formed by the angles of the ribs. In other cases, again, in which the collapse is local only, the affected areas are distinguished from the rest of the organ by being depressed, of darker colour, limp and tough to the feel, and easily sinking in water.

Symptoms resemble in great measure those observed in pleural effusion. The patient complains of dyspnoea; he is livid; the intercostal spaces are drawn in at each laboured inspiration; and there may be more or less pain over the affected side of the thorax.

The signs which this condition give rise to are, a feeble and distant respiratory murmur, with brouchophony over the situations of the larger bronchial tubes. Occasionally a fine crackle is heard, especially at the edges of the lung, at the end of a deep inspiration. This is doubtless due to distension by a forced inspiration of certain of the air-vesicles, which are not usually stretched, and in which, consequently, a certain amount of mucus collects. But we constantly detect the same sign in the lungs of people whose occupations confine them to close and ill-ventilated rooms, and in whom no other sign of partial collapse of lung is evident. The above interpretation, however, is probably a correct one.

In children the signs of pulmonary collapse, in the main,

resemble those of consolidation of the lung or of a well-defined thoracic tumour.

Treatment.—In congenital atelectasis the main indications of treatment are properly arranged passive movements of the chest:—really a form of gymnastics, with a view to expand the lungs.

In the collapse due to pressure on the pleural surface of the lung it is necessary first to remove the fluid or growth if possible. Afterwards the lung may show signs of again being distensible. This should be facilitated by gentle muscular exercises of the chest. Aspiration has been successfully practised in cases of collapse with pneumo-thorax.

DISEASES OF THE PLEURA

PLEURISY (PLEURITIS)

Definition.—An inflammation of one or both pleuræ. The disease may be acute, sub-acute, or chronic.

Acute Pleurisy

Causation.—(1) PRIMARY.—The disease may be due to exposure to cold, wet ; or it may be induced by blows on, or injuries to, the chest wall. Few people pass through life without a milder or severer form of pleurisy ; but the affection is more frequent in *youth* and probably in *males*, owing to the greater exposure to cold and injuries.

(2) SECONDARY.—It may be secondary to acute specific fevers (scarlet, typhus), to acute rheumatism, or to albuminuria. It not infrequently results from septic poisoning (pyæmia, septicæmia) ; also from new growths in the pleura or on the surface of the lung (tubercle, carcinoma, sarcoma). Finally it may be due to extensions of inflammatory infiltration from the lung, pericardium, liver, or mamma.

In many instances, however, it is difficult to determine whether the condition be primary or secondary.

Pathology.—(1) The onset is marked by hyperæmia of the capillary vessels of the pleura and subpleural connective tissue. (2) Then ensues proliferation of the epithelium and connective-tissue elements, so that the surface of the membrane becomes dull and sticky. (3) Exudation of coagulable lymph containing fibrin and inflammatory cells, the

latter being derived from the proliferated epithelium of the part affected and from the migrated leucocytes. The effused lymph in the most acute cases is sufficient to obliterate the pleural cavity at the site of the inflammation and agglutinate the two layers of pleura. The inflammatory membrane thus formed may vary from a thin lamella to the thickness of half an inch ; it may also, as in pericarditis, present a ribbed, villous, or reticulated aspect.

The subsequent effects of the inflammatory process are the appearance of a newly-formed membrane of connective-tissue bands running from visceral to parietal layers. These ultimately become either organised and contain capillary blood-vessels, or are absorbed and disappear during convalescence. The inflammatory process is not always limited to the area of onset, but may invade the neighbouring lobe of lung, or the pericardium, or the upper surface of diaphragm.

In the sub-acute cases serous effusion is a more marked condition, whilst the formation of lymph is less pronounced, only showing itself as patches on the surface of the pleura, or as detached flakes floating in the serous fluid. The amount of fluid may vary from an inappreciable quantity, producing no mechanical effects, to four or five pints or even more, with resultant pressure upon, and dislocation of, neighbouring vessels, tubes, and organs. Hence the heart may be pushed over the mid-line of the body ; the diaphragm, with the liver, spleen, or stomach, may be depressed ; the large vessels in the upper part of the thorax rendered turgid ; whilst the chest wall is bulged outwards, and the lung itself compressed towards the posterior mediastinum.

Subsequently one of three events occurs : either (1) the effused fluid causes death by its mechanical embarrassment of heart or lungs ; or (2) it becomes absorbed and entirely disappears, leaving only the fibrinous elements on the surfaces of the pleura from which adhesions generally form ; or (3) degenerative changes occur in the inflammatory products, and the formation of pus takes place. The pleural cavity then is, to all effects, an abscess cavity, which may point anywhere, following the course of the least resistance. Thus it may,

unless relieved by surgery, discharge itself through the chest wall, or through the diaphragm, or into the pericardium, or into the lung itself.

As regards the adhesions which form, especially as the result of chronic pleurisy, they may, as stated above, vary in thickness ; they may also remain as tough, fibrous bands, or become converted into a cartilaginous-like structure, infiltrating the interlobular septa of the lungs, and not infrequently undergoing calcareous degeneration. Any area of the pleura may be affected from diaphragmatic surface to summit.

Symptoms.—The onset is marked by sudden febrility, the temperature rising to 101° perhaps in twenty-four hours ; but in uncomplicated cases it rarely attains so high a point as in pneumonia, and follows no regular course. The bowels are usually constipated, the tongue is furred, and there is more or less thirst and anorexia. *Dyspnœa* comes on early in the disease, the respiration being quick and shallow, or ‘catching,’ especially at first. There is usually some *cough*, but it is not so urgent a symptom as in pneumonia, and is unattended by expectoration. *Pain* is the most pronounced and earliest symptom : it assumes the character of acute, lancinating ‘stitch,’ limited to the affected side, and increased by cough, respiration, and other thoracic movements. The *position* of the patient varies according to the stage of the disease : in the early stage, when there is much pain, he seldom lies on the affected side, as the pressure increases the pain. His appearance is anxious ; but his face is not congested as in pneumonia, although it may be livid from compression of the vena cava. Delirium and subsultus and the typhoid state may occur as in pneumonia, but are comparatively rare symptoms. Towards the fourth or fifth day, when effusion occurs, the dyspnœa somewhat subsides, the pain is less severe, and he now lies on the affected side so as to give the opposite healthy lung full play.

Physical Signs.—(a) BEFORE EFFUSION occurs the earliest signs are imperfect chest expansion, followed by pleuritic friction and occasionally friction fremitus, although the latter is more common when the inflammation is clearing up. The percussion note is unaltered.

(b) AFTER EFFUSION.—The breath sounds are usually diminished and distant, but they may become tubular if the lung be compressed. Vocal resonance over the dull area is diminished or absent, whilst at its upper limit it is modified into ægophony. Vocal fremitus is also diminished or absent—a most valuable sign. Dulness over the affected area is marked, percussion producing a resistant feeling, and a ‘tubby’ note which may extend over the mid-line of the body. With much effusion there is obliteration of the intercostal spaces, and pain is diminished. There is also displacement of viscera : thus if the left side be affected the heart may be pushed over to the right, and be found beating almost in the right axilla, and the spleen displaced downwards ; whilst if the right side be involved, the liver is depressed downwards and slightly towards the middle line. If the effusion be excessive, lividity may occur from pressure on the trunk veins, but usually it exerts no permanent injurious effect upon the œsophagus or bronchi. As the fluid becomes absorbed, and the two surfaces of pleura again come together, a coarse, grating, sound is heard (*redux crepitation*), which is often attended by friction fremitus and, it may be, by a return of pain.

Complications.—(1) *HYPER-PYREXIA*, accompanied by delirium, more especially if the inflammation be excessive, or extend to the pericardium. (2) *PRESSURE* upon, and displacement of, neighbouring organs. (3) *HÆMORRHAGE* into the pleura. (4) *EMPYEMA*, with possible resultant pyæmia, caries of ribs, and lardaceous degenerations.

Prognosis.—Favourable in simple uncomplicated cases ; but it is often a grave condition if secondary to phthisis or other disease of lungs ; or if it supervene on pericarditis ; or if it result from septicæmia and other forms of blood disease, the exudation in all these cases soon becoming converted into pus.

Treatment.—The patient’s room should be warm, between 60° and 70° F. The first indications are, to ensure as much rest as possible to the inflamed side, and to relieve the pain. The application of stout strapping or a plaster-of-Paris jacket

has been advocated ; the objections to these procedures are the difficulty they present to exploration and examination of the chest. Opium or hypodermic injection of morphia may be given somewhat freely. Leeches often relieve the early pain, but should only be applied to robust, plethoric patients. A preliminary purge is generally advisable. The diet should be light and nourishing, but with slightly restricted quantity of fluids.

During the stage of effusion, and when the fever has subsided, we may apply a series of flying blisters, and paint the side freely with iodine liniment.

If the effusion does not clear up, or tends to re-accumulate after partial absorption, we may resort to diuretics or to watery purges (magnes. sulph. gr. xxx. t. d.) with good results. But if embarrassment of respiration or of circulation be serious ; if the heart be displaced ; or if the fluid fills one side of the chest, or remains stationary for any length of time, such as two weeks, it is best not to delay, but to perform *paracentesis thoracis*. It is remarkable, however, what a large quantity of effusion can be tolerated in the chest, often giving rise to no symptoms of discomfort whatever. The question of how much fluid should be removed, is often raised. In our experience it is best to remove as much as possible. Iodide of potassium is a valuable adjuvant to aid absorption of remaining fluid.

Sub-acute Pleurisy

Definition.—A low, inflammatory affection of the pleura, often accompanied by much serous effusion.

Causation.—It generally occurs in weak and feeble patients, and is often secondary to some general disease, *e.g.* phthisis, or Bright's disease. Or it may be caused by fatigue or exposure.

Symptoms may be so slight as to be overlooked. Pain and fever are usually slight, and, until the effusion be pronounced, cough and dyspnoea are not severe. The pulse is quick and feeble, and often there are excessive night sweats. The condition is seldom ushered in by chill or rigor.

Physical Signs are the same as in acute pleurisy with effusion.

Treatment.—Avoid depressing remedies. A series of blisters may be applied to the affected side. The food should be nourishing, yet with diminished quantity of fluids. Iron tonics are usually indicated. Iodide of potassium is a valuable remedy to aid in absorption of the fluid.¹ If the fluid does not diminish after two weeks' treatment, perform aspiration. The usual indications for paracentesis are : (1) excessive amount of effusion ; (2) if the effusion remain stationary ; (3) dyspnœa, or cardiac failure ; (4) hectic fever.

Chronic Pleurisy

Definition.—An inflammatory affection of the pleura, running a chronic course, and causing fibroid thickening of the membrane.

Causation.—(1) A previous acute attack ; (2) tubercular or malignant infiltration of the pleura. It is more likely to occur in thin, debilitated subjects with defective chest movements.

Pathology.—The pathology of the early stages is the same as in acute pleurisy. Absorption, however, does not completely take place ; and as a result of the chronic irritation of the pleural membranes, they become thickened and indurated, the changes being partly due to layers of superimposed lymph, and partly to the proliferated fibrous and connective-tissue elements of the pleura. This thickening may vary in extent from a cardboard-like layer to one measuring an inch or more across. Subsequently it may extend into the lung tissue, selecting, as it were, certain paths between lobules where connective tissue is abundant, until the lung is freely infiltrated with fibrous bands and partly destroyed.

Symptoms, and Physical Signs.—Cough, dyspnœa, retraction of affected side of chest, with loss or impairment of respiratory movement. Together with these one would

¹ ℞. Potassii Iodidi gr. v. ; Magnes : Sulphatis gr. xx. ; Aquæ Camph : ad ʒj. Misce.

expect to find persistent dulness, but without the modifications of breath sounds and vocal phenomena present in effusion or consolidation. Nor would there be the intermittent temperature significant of empyema. Creakings and other adventitious respiratory sounds are common.

Treatment consists entirely of remedial measures to allay cough and dyspnœa. Frequent blisters, or the application of iodine to the affected side, may retard the disease and lessen the tendency to fibrous thickening of the pleura.

EMPYEMA (PYO-THORAX)

Definition.—The formation of pus in the pleural cavity. It may run an acute or a chronic course.

Causation.—It is rarely a primary affection; but is generally secondary to (1) acute pleurisy, or (2) some condition or state of patient, such as an attack of an acute specific fever, determining the formation of pus.

Pathology.—It is one of the terminations of an acute attack. The plastic exudation becomes purulent, and the inflamed surface of pleura becomes to all intents a pyogenic membrane.

Symptoms, and Physical Signs.—Hectic, with rigors, night sweats and intermittent temperature occurring in a patient in the third or fourth week of an attack of pleurisy would make one suspicious of empyema. Besides these symptoms we should find bulging, redness, and œdema of the intercostal spaces, with absolute dulness and total absence of breath and voice sounds, the higher harmonics of the vocal note even being absorbed. But this last sign is by no means reliable.

Prognosis.—Formerly unfavourable or doubtful; but results have been greatly improved by strict antiseptic precautions when evacuating the pus. The dangers to be apprehended are, exhaustion, with diarrhœa; amyloid degeneration of the liver and other organs; and pyæmia.

Treatment.—An exploratory puncture with a grooved needle or a hypodermic syringe may be made first. If pus be detected it should be evacuated without delay. This may be

done by repeated aspirations, or by free incisions into a carefully selected intercostal space, and the insertion of a drainage tube, so as to allow of the free discharge of the pus into antiseptic dressings. The discharge of pus may be facilitated by irrigation of the cavity by some antiseptic fluid. The dressings should be frequently changed during the first four days after the operation. If the intercostal spaces be contracted by overlapping ribs, resection of a portion of rib may be required. Cleanliness and antiseptic precautions are essential.

If the abscess be left alone, it usually discharges itself either into a neighbouring bronchus, or externally, or into some near cavity or viscus, with disastrous results. Rarely, the abscess dries up and becomes converted into a cheesy or calcareous mass. The patient's strength must be maintained by stimulating and nutritious food, and tonics.

HYDRO-THORAX (DROPSY OF THE CHEST)

Definition.—A collection of serous fluid in one or both pleural cavities.

Causation.—The condition is always a secondary one, dependent on disease of the heart, lungs, kidneys, or the blood itself.

Pathology is that of dropsy. The accumulation of fluid may be due to some mechanical obstruction to the flow of blood in one or other part of the circulation ; or it may be caused by some inflammatory exudation ; or result from obstruction to the lymph vessels. Given a passive congestion of the capillaries of the lungs, a clear, non-inflammatory exudation is poured either into the air-cells (pulmonary œdema), or into the pleural cavity constituting hydro-thorax. The amount of effusion may vary from a few ounces to three or four quarts ; it may increase suddenly and rapidly ; and the quantity may be augmented after death, it being a common observation that the amount of effusion in the pleura at the autopsy is often altogether out of proportion to the physical signs noted during life.

Symptoms, and Physical Signs.—There is usually some

associated œdema in some other organ or tissue, together with dyspnœa, lividity, and slight cough, but with little or no fever. The physical signs are mainly those of effusion, viz. : dulness in lower part of the chest, ægophony, distant breath sounds, and obliteration of intercostal spaces ; *both* sides are usually affected, thus distinguishing the condition from pleurisy.

In persistent effusion into the pleuræ, which is often a result of Bright's disease, the urine should be examined carefully and repeatedly for albumin.

Treatment.—Aspirate the fluid, in whole or in part, especially if the dyspnœa be urgent. Then direct measures to alleviate the disease of which it is a symptom. Watery purgatives (pulv. jalapæ co.) may be given daily ; elaterium (gr. $\frac{1}{16}$) is also useful, unless the patient be too debilitated. Digitalis is indicated when cardiac failure is the primary cause.

PNEUMO-THORAX

Definition.—The presence of air in the pleural cavity.

Causation.—Usually due to the extension of tubercular cavity from the lung into the pleural sac. It may also result from surgical operations on the chest walls or about the root of the neck. Rarely as a result of rupture of distended air-vesicles in emphysema. It may also be caused by violent injuries to chest wall, or by the discharge of localised empyema either through the parietes, or through the lung itself.

Pathology.—In pulmonary phthisis, running an acute course, a tubercular mass near the surface of the lung may rapidly break down, and so admit air to the pleura before adhesive pleurisy has occurred. This is a common cause of pneumo-thorax occurring in the early stages of consumption. The perforation may take place at any part of the lung surface, and it is not infrequently valvular in character.

The effects of the admission of air to the pleural cavity are usually (1) acute pleurisy, and (2) empyema.

Symptoms, and Physical Signs.—The symptoms are pronounced. They consist of sudden pain, dyspnœa, with pale,

dusky face, quick and thin pulse, and all the conditions known as shock. The affected side is enlarged, the intercostal spaces are effaced, and there is no movement attending inspiration. Percussion note is highly tympanitic, and it is found that the heart has been pushed over to the healthy side, whilst the liver or spleen is depressed.

On auscultation the respiratory murmur is very distant or totally absent, whilst also there is complete absence of vocal fremitus and resonance. The chest is practically a large cavern, and thus produces all the modifications of respiratory and percussion sounds—such as amphoric breathing and *bruit d'airain*—common thereto.

After a short but variable time the signs of pleurisy with serous effusion are superadded. Succussion splash is then a typical and diagnostic physical sign.

Treatment can only be palliative in most cases. Give opiates, or chloroform inhalation, to relieve the pain.

Aspiration may be resorted to if pain and dyspnœa be urgent, but the relief is temporary only, unless it be in those cases which are produced by external injury, or in which the valvular opening from the lung has healed up.

DISEASES OF THE VASCULAR SYSTEM

ANATOMY OF THE HEART

The **Heart** weighs in the adult from nine to ten ounces, being slightly smaller in women.

It occupies the middle mediastinum, and is enclosed in a fibrous bag, or pericardium, which is attached below to the central tendon of the diaphragm, but above is continuous with the external coats of the aorta, pulmonary artery, superior cava, and large vessels, and is ultimately connected with the deep fascia of the neck. This bag is again lined by a smooth serous sac, which, extending along the aorta and pulmonary artery for about two inches and enclosing them in a common tube, is reflected downwards and invests the entire outer surface of the heart.

The heart as it lies in the thorax is partly covered by the lungs; the greater portion of the right ventricle, however, is exposed; and the left ventricle is situated posteriorly and to the left, resting on the diaphragm.

The absolute dulness of the heart may be represented by a circle of one inch radius, the centre of which is midway between the left nipple and the lower end of the sternum. (Holden.)

In a mesial section of the body, about three-fifths of the heart are to the left side, and two-fifths to the right.

The relations of the heart to the chest walls, and the relative positions of the valves, are described under Physical Examination of the Heart (Auscultation).

The capacities of the four cavities of the heart are about

equal, holding from five to six ounces of blood apiece. The right cavities, however, may be slightly in excess of this measurement, owing to the thinness of their walls, as compared with the left side.

The walls of the left ventricle are thicker than those of the right, in the proportion of three to one. They are on both sides, thickest at about midway between apex and auriculo-ventricular septum.

The circumferences of the various orifices are as follows : aortic, $3\frac{1}{4}$ in. ; pulmonic, $3\frac{1}{2}$ in. ; mitral, 4 in. ; tricuspid, $4\frac{1}{3}$ in.

Roughly speaking, the mitral orifice will admit two, whilst the tricuspid will admit three fingers.

The **Arch of the Aorta** is divided into three parts : an ascending, an antero-posterior or transverse, and a descending portion.

The first portion passes upwards, and to the right, to a quarter of an inch beyond the sternum, behind the second and the first interspaces. Its relations consist mainly of vessels which contain venous blood. In front are the pulmonary artery and right auricular appendix. Behind are the right division of the pulmonary artery and the root of the right lung. To the right we find the superior vena cava and the right auricle ; and to the left is the main trunk of the pulmonary artery.

The second portion runs beneath the manubrium of the sternum, immediately above its junction with the gladiolus.

Above are the innominate, the left carotid, the left subclavian arteries, and the left innominate vein.

Below, it is in relation with the bifurcation of the pulmonary artery, the ductus arteriosus, the left recurrent laryngeal nerve, and the left bronchus.

In front are the left vagus and phrenic nerves, the superficial cardiac branches of the sympathetic, the left superior intercostal vein, and perhaps the left innominate vein, if the vessel is lower than usual. It is also overlapped by the left lung and pleura.

Behind are the trachea and œsophagus, the left recurrent laryngeal nerve, the thoracic duct, and the deep cardiac plexus.

One or two lymph glands are contained in the concavity of the arch.

The third portion lies on the backbone. It has the pleura externally, the œsophagus internally, and the root of the left lung in front.

A knowledge of the **Development** of the heart is important in order to understand the existence of malformations. The heart is developed from the cells of the mesoblast, which forms at an early period of foetal life two tubes, one on either side of the body. Each tube receives posteriorly a large vessel or vein, and becomes prolonged anteriorly into an artery. As these two tubes coalesce in the median line, a single central tube is ultimately formed, with consequently two veins entering behind, and two arteries issuing in front. This tube eventually becomes elongated and bent on itself, so as to become U-shaped, and is subsequently divided by two transverse constrictions into three parts : one of which develops into the aortic bulb, another into the auricles, and the third into the ventricles. Another subdivision of each of these cavities again takes place, and hence are formed the aorta and pulmonary artery, the right and left auricle, and the right and left ventricle, respectively.

The septum which separates the ventricles commences at the apex and extends upwards ; whilst the septum dividing the auricles is developed as a fold, which extends from above downwards.

PHYSICAL EXAMINATION OF THE HEART

In the physical examination of the heart, evidence is obtained by Inspection, Palpation, Percussion, and Auscultation. We may, if we wish, supplement these processes by the cardiograph and other physiological instruments.

Inspection.—The patient should, if possible, have the chest thoroughly exposed. The situation of the apex-, or rather impulse-beat should be carefully noted. Ordinarily it can be easily detected, and is found in normal hearts to be situated one inch below and half an inch internal to the left nipple. Occasionally, in perfect health even, the impulse is not seen

or it may be so diffused as to constitute a difficulty in exactly localising it. On the other hand, the impulse may be wanting in weakened conditions of the heart's muscle, or in debilitated subjects. Or it may be displaced, downwards and outwards in cardiac hypertrophy, upwards in pericardial effusions; or it may be detected in quite an abnormal locality of the chest, from pleural effusion, from encroachments of neighbouring organs or new growths, and (rarely) in transposition of viscera.

'Normal impulse' is an indefinite term. It can only be appreciated by constant observation, and the comparison between healthy and unhealthy hearts. Excessive impulse is usually associated with hypertrophy of one or other or both sides of the heart, and is then not infrequently accompanied by some bulging of the precordial region. It is also seen in people of nervous temperament.

Normal rapidity of the heart's action corresponds to the rate of the pulse-beat, and varies with age, sex, stature, temperament, occupation, &c. The rapidity is usually between seventy and eighty beats per minute, being somewhat more frequent in females than in males; whilst in children it amounts to one hundred beats, and upwards. Excessive rapidity, amounting to two hundred beats or even more, is known as *tachycardia*; and diminished frequency, sixty or even less, constitutes *bradycardia*.

The variations in frequency of the heart's action are more fully considered under the heading 'Pulse.'

Palpation.—It is necessary not only to see where the heart's impulse is, but also to fix its exact position by placing the hand over the pulsating area, and, if possible, marking the site of its greatest intensity. We thus obtain more perfect knowledge as to the heart's situation, condition, and its rapidity of action.

The impulse may be increased in violent nervous action and in hypertrophy of the heart's walls; or it may be decreased in collapse, in cardiac atrophy, in pericardial effusions or adhesions, and by the interposition of overdistended lungs.

Besides these conditions we may by palpation detect the

thrill common in mitral stenosis, aneurysm, and septal imperfections; or *valve tremors*, as when a segment or flap of a valve is lacerated or grossly incompetent; or, again, a *friction fremitus* occasionally in a very roughened pericardium. By palpation we also take notice of the heart's rapidity of action; or of its rhythm, in such cases where the pulse does not reach the wrist (dropped beats), or when, from whatever cause, the radial pulse cannot be taken. Finally, marked displacements of the heart may be felt as well as seen.

By **Percussion** we are enabled to map out the heart's area of dulness. It should extend as high as the level of the upper border of the third rib; its lower boundary would be marked by a line from the end of the xiphoid cartilage to the apex-beat; the left boundary would follow a line from about the middle of the second cartilage on the left side, downwards and outwards to the apex; whilst the right-hand limit of dulness should reach only as far as the mid-line of the sternum. The area of absolute cardiac dulness is even much more limited than this; on the other hand, the deep cardiac dulness may be made out to extend almost as far as the heart's limits themselves.

An *increased cardiac dulness* may be brought about by hypertrophy of one or other of its cavities, or by dilatation thereof; and also by aneurysm of its walls. It may also be produced by retraction of lung, secondary to cavity or adhesions, a greater area of heart being thus left uncovered. Or it may be a physical sign of pericardial effusion, or of new growth.

A *diminished cardiac dulness* is found in atrophy of the organ, or it may be caused by the extensive overlapping of emphysematous lung, or the intrusion of air into the pleura.

As in percussion of the lungs, so in percussion of the heart, much valuable information is gained by the sense of resistance which is afforded by pericardial fluid of great tension, and by excessive hypertrophy.

Auscultation.—It is necessary to auscultate all the valves of the heart, and also the walls of its various cavities.

(a) **Normal Sounds.**—The two sides of the heart should act in unison, and should produce by the contraction and

the dilatation of the ventricles two separate and distinct sounds, followed by a pause. The first sound is best heard at the apex : it begins abruptly, increases somewhat in volume, and then gradually diminishes. It is caused by the contraction of the ventricle walls, by the rush of blood through the infundibula, by the opening of semilunar valves, by the tension of auriculo-ventricular valves, and by the impact of the heart against the chest wall. This first sound is followed by a short, sharp, second sound, heard best at the base, and especially over the aortic valve to the right of the sternum. It corresponds with the closure of the aortic and pulmonary valves, and with the diastole of the ventricles, the former being the chief factor in its production.

This is followed by a pause or interval, which is again succeeded by the first sound.

The two sounds thus heard have been variously imitated.

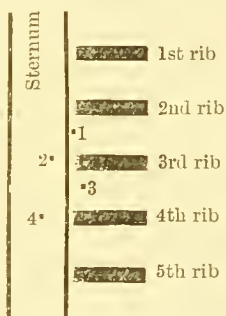


FIG. 13.—DIAGRAM TO REPRESENT THE POSITION OF VALVES

Perhaps the most convenient and apt compound word which will represent these sounds is '*one-two, one-two*,' the accent occurring on the first sound at the apex, and on the second sound at the base. (Broadbent.)

The relation of heart valves to the front of the chest is as follows :

(1) The pulmonic is situated at the left-hand border of the sternum, in the second interspace.

(2) The aortic is behind the sternum, but a little to the left of the mid-sternal line, and on a level with the third costal cartilage.

(3) The mitral is beneath the third interspace, just external to the left border of the sternum.

(4) The tricuspid is on a level with the fourth cartilages, and is bisected by the mid-sternal line.

The pulmonic is most superficial, whilst the mitral is deepest.

In addition, it is necessary to observe whether the heart maintains its normal regularity, rhythm, and rapidity, as any deviation from the normal standard is significant of disordered innervation, or of altered structure, or other form of disease.

(b) **Abnormal Sounds of Heart.**—As regards alteration in rhythm, *reduplications* of the first sound heard at the apex, or of the second sound heard at the base, are not infrequent. They may occur in people apparently healthy ; but they are usually associated with some disturbance of the systemic or pulmonary circulation, and with the consequent hypertrophy of one or other side of the heart. The reduplication of the second sound is obviously due to extra tension in the left system, as compared with the right, or *vice versa*. The causes of reduplication of the first sounds are not at present quite so clear. These reduplications can also be felt by an educated finger. *Prolongation* of first sound, most frequently heard at the apex, frequently presages early valve disease, and is of some importance in diagnosis of an oncoming lesion before actual murmur is detected.

Accentuation may occur at the mitral orifice, with systole (the 'snap' in mitral stenosis); and with diastole at the aortic valve when associated with high arterial tension (Bright's disease, &c.), or at the pulmonic valve, as evidence of tension in the pulmonary circulation (chronic lung disease, mitral disease, &c.).

Diminution of heart sounds occurs in weak action of the heart. Or it may be due to muffling of its sounds by an overlapping of emphysematous lung, or by pericardial effusion.

Irregularity and *intermission* of beat are found in structural disease of heart walls or valves. They may be evidences of disordered innervation ; but they are frequently consequent on

dyspepsia, and other stomach derangements such as result from the use of strong tea, tobacco, and other poisons.

Both are occasionally observed in people who have no other symptoms of disease of the vascular apparatus. Our experience leads us to think that, apart from disease of the central organ, intermission is very often observed in persons of tall stature, and especially in men who have chronic disease of bladder, prostate, or urethra.

It is necessary to observe that, in some cases, whilst the pulse is intermittent, the heart is only irregular ; that is to say, a pulse-wave, owing to a weak ventricular stroke, may not reach the wrist, thus giving rise to an apparent intermission which does not really exist. This condition is known as 'dropped beat.'

Murmurs may be divided into two great classes, viz. : organic, which include endocardial and pericardial ; and inorganic, or so-called hæmic or functional murmurs. Cardiac murmurs of the first class, therefore, indicate structural change.

The endocardial murmurs vary in character from a gentle blowing sound to a harsh rasp or even loud musical tone. It is useless, however, attempting to define them, as, after all, their musical character is seldom of great importance, and a given murmur may vary considerably under different circumstances, as, for example, during repose and excitement.

The pathological conditions which cause endocardial murmurs may be briefly summarised as structural changes, congenital or acquired, which give rise to obstruction to, or allow of regurgitation of, the blood stream. These changes may be in the valve flaps, or in the orifices, or in both together, and for the most part are of inflammatory origin.

It may perhaps be affirmed that, as a rule, the louder the murmur, the greater is the organic change. Further, although the diagnosis of the site of the murmur does not by any means depend on auscultatory signs alone, the rule generally obtains that the murmur is of greatest intensity over the valve at which it is generated. We thus listen over those chest 'landmarks' which indicate the situations of the valves (see fig. 13). But this is not sufficient for diagnosis, seeing

that all the cardiac valves are so close together that they could be overlapped by a crown piece. We therefore seek for additional auscultatory aid by determining in which direction the blood stream, and therefore the sound, is propagated—whether it be upwards to the right, as in aortic obstruction; or downwards to the xiphoid cartilage, as in aortic regurgitation; or round to the left axilla, as in mitral regurgitation. It may, in addition, be stated that probably over ninety per cent. of endocardial murmurs are due to disease of the valves (aortic and mitral) on the left side of the heart.

The importance of detecting heart-murmurs cannot well

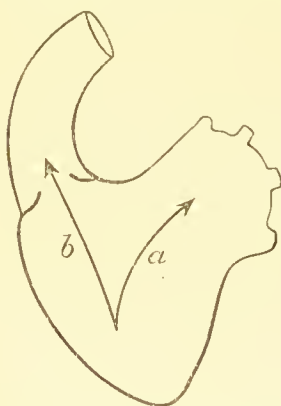


FIG. 14.—SYSTOLIC MURMURS
a, Mitral regurgitant; *b*, aortic obstructive.

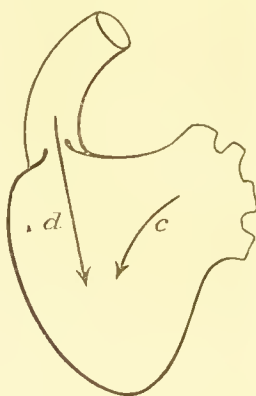


FIG. 15.—DIASTOLIC MURMURS
c, Mitral obstructive; *d*, aortic regurgitant.

be overestimated, as they form the most conclusive evidence as to the existence of valvular disease.

A. When organic murmurs are generated in the left heart, we seek to ascertain the following clinical data, viz. :

- (1) The situation of the greatest intensity of the murmur, *i.e.* whether it is loudest at apex or base.
- (2) Whether the murmur accompanies or replaces the first or the second sound of the heart.

Murmurs heard best at the apex are produced at the mitral orifice; whilst those which are loudest at the base are produced at the aortic orifice. It follows, therefore, that an apical murmur

accompanying systole is significant of mitral regurgitation (fig. 14, *a*) ; if it occur during the diastolic period it is diagnostic of mitral obstruction (fig. 15, *c*). Similarly, with a basic murmur attending systole we infer the existence of aortic obstruction (fig. 14, *b*) ; whilst if it replace the second sound it implies the presence of aortic regurgitation (fig. 15, *d*). (See table, p. 404)

Beyond this, each murmur has special and peculiar qualities of its own which are discussed further on.

Apical or Mitral Murmurs.—MITRAL REGURGITATION is the most frequent, and is probably met with oftener than all the other valvular murmurs together. The murmur of this lesion attends the first sound, is loudest at the apex, and is conducted towards the axilla, *i.e.* in the direction of the reflux blood stream. In the majority of cases it is said to be also easily detected at the lower angle of the left scapula. Its character varies : it is usually soft and blowing, but may be harsh and grating, or musical, or attended by a vibratile purr. Compensatory hypertrophy of the left ventricle occurs sooner or later.

MITRAL OBSTRUCTION.—This condition is common in children and early adolescents. The murmur is localised, and best heard a little above the apex-beat ; it may occupy the whole of the diastolic period, or only part of it. If it fill the whole of the diastolic period, it is heard beginning immediately after the termination of systole, and fills up the time occupied by the second sound and the period of rest. The murmur may be loudest at the commencement, at the middle, or at the termination of the diastolic period ; or it may be heard only during one of these fragments of the diastolic period, and is then spoken of as the early, the middle, and the late diastolic (presystolic) murmur, as the case may be. The presystolic, occurring at the time of the auricular systole, is the one most usually heard.¹

These murmurs are generally of a loud character, but of limited area ; and if they run up to systole they terminate in a well-marked click or 'snap.' They are often described as whizzing, purring, or grinding. They are usually attended by

¹ Bristowe : *Med. Soc. Proc.* vol. xi.

an easily perceptible thrill. They may disappear temporarily after prolonged rest, and are perhaps, therefore, the most inconstant of all organic murmurs, varying according to the amount of obstruction and the force of the current. It is well to remember that mitral obstruction may exist without murmur when the left auricle is failing.

As a result of the obstruction, hypertrophy and dilatation of the left auricle occur, together with accentuation and reduplication of the second sound over the pulmonic valve.

Basic or Aortic Murmurs.—AORTIC OBSTRUCTION is more common than aortic regurgitation, and probably ranks in frequency next to mitral regurgitation. It is heard loudest over the right third cartilage, *i.e.* near the valve, and it may be traced over the arch of the aorta. The murmur varies according to the force of the ventricle stroke, and the character and amount of the obstruction. It is generally, however, of a harsh, blowing nature, and usually occupies the whole of the period of the first sound. At times an aortic systolic murmur may be detected towards the end of the systolic period only, suggesting that the roughened obstruction is situated in the aorta itself, at some little distance from the valve.

Hypertrophy of the left ventricle occurs as a sequel if the obstruction be marked ; but slight obstruction may undoubtedly exist without hypertrophy. It is also probable that in all cases of aortic obstruction a certain degree of regurgitation occurs as well.

AORTIC REGURGITATION is usually heard best over the third right costal cartilage, or in the middle of the sternum. It may at times be most intense over the left second cartilage, or even at the lower end of the sternum. Occasionally it seems propagated towards the apex. It accompanies part of the second sound, or wholly replaces it. The murmur is usually a soft one, with characteristic bellows or 'blast-furnace' tone which is quite characteristic.

B. Organic murmurs produced in the right side of the heart are in connection with either the pulmonic or the tricuspid valves. They are very rare in comparison with left-sided murmurs.

In diagnosis we are guided by rules similar to those which we observe in examining the left side of the heart. (See table, p. 404.)

Apical or Tricuspid Murmurs.—**TRICUSPID REGURGITATION** is rare as a primary valve lesion, and is usually secondary to disease of the left heart or to obstruction in the pulmonary circulation. The great majority of cases have been observed in females, or in persons suffering from displacement of the heart. The murmur is heard with systole, over the right side of the sternum, at about the level of the fifth costal cartilage. It is a soft, low, blowing murmur, propagated to the right border of the heart, whilst it is inaudible at the apex-beat.

TRICUSPID OBSTRUCTION.—A murmur of extreme rarity is heard during the diastolic period, immediately before systole, in the fourth space close to the right border of the sternum. The most frequent cause is some congenital defect.

Basic or Pulmonic Murmurs.—**PULMONIC OBSTRUCTION** is generally due to some congenital defect (stenosis, or intra-uterine endocarditis). The murmur attends the heart's systole, and is of varying degrees of intensity, according to the amount of obstruction and the force of the ventricle stroke. It is loudest over the second left interspace close to the border of the sternum. The diagnosis would be confirmed by general cyanosis with coldness of the extremities and clubbing of the fingers.

PULMONIC REGURGITATION is also extremely rare. It may be caused by some congenital defect of valve, or by a general endocarditis affecting this as well as the other heart valves. The murmur occurs with diastole, and is best heard over the third left costal cartilage at its junction with the sternum; thence it may be propagated down the right ventricle to the apex. It is neither so loud nor so prolonged as the aortic regurgitant murmur.

Multiple Endocardial Murmurs are frequent, and their differential diagnosis will depend on their sites, physical characters as described above, together with other concomitant signs.

Thus aortic regurgitation is often associated with aortic

obstruction, or with mitral regurgitation. Mitral regurgitation and obstruction are also frequently met with, and indeed a double murmur at both orifices in the left ventricle may exist.

Again, tricuspid regurgitation may supervene as a secondary complication to valvular disease of the left side.

Pericardial Murmurs, or Friction Sounds, are due to inflammatory affection of the pericardium, producing thickened and roughened surfaces which normally are smooth. We thus have a to-and-fro murmur over the cardiac area, the component parts of which are synchronous with systole and diastole. The murmurs, if not very intense, bear a resemblance to those heard in double aortic disease. But they are generally rougher in tone, and apparently more superficial. They are increased by pressure of the stethoscope, and are most marked over the right ventricle, this being due to the stronger left ventricle pushing the exuded lymph over to the right (Peacock), or possibly to the rotation of the heart on its long axis during systole. The murmurs are often attended by a perceptible thrill or fremitus ; they may disappear when much fluid is exuded, to reappear when the serum is absorbed.

Inorganic or Hæmic Murmurs.—The causes of these murmurs are various. (1) They may result from some faulty condition of the blood, as in chlorotic girls or anæmic males ; (2) from some irregular or excessive contraction of the muscular apparatus of the heart ; or (3) from some abnormal tension of the larger vessels. In the first category the murmur is most frequently heard over the situation of the pulmonic valve ; it is generally systolic in period, and of a soft blowing character ; but indeed it may be harsh and loud, and heard all over the cardiac area, and so simulate an organic murmur.

The second variety is occasionally heard in cases of violent palpitation attended by high arterial tension, or in disordered and irregular contraction of heart walls and *musculi papillares*. It is most marked over the aortic valve and in the course of the aortic arch. Neither form is attended by cardiac hypertrophy, unless in the later stages, after prolonged palpitation.

TABLE I

Normal		Abnormal	
		<i>Condition</i>	<i>Significance</i>
Inspection	Normal site of apex-beat	Diffused . . .	Weak systole
		Displaced . . .	Pericardial effusion
		Transposed . . .	Congenital malformation
		Increased . . .	Cardiac hypertrophy
		Diminished . . .	Cardiac atrophy
Palpation	Normal impulse	Bulging . . .	Pericardial adhesion; aneurysm
		Excessive rapidity (tachycardia)	Neuroses; fevers; valve lesions; disease of muscular walls
		Diminished rapidity (bradycardia)	Disease of central nervous system; exhausting diseases
		Increased impulse	Cardiac hypertrophy
		Diminished impulse	Cardiac atrophy
Percussion	Normal limits of dullness	Thrill . . .	Stenosis of orifice; sacculated aneurysm of the arch
		Valve tremors . .	Ruptured valve
		Friction fremitus .	Pericardial friction
		Dropped beats . .	Weakness of ventricular systole, or disordered nervous apparatus
		Increased dullness .	Hypertrophy; dilatation; aneurysm; retracted lung; pericardial effusion
Auscultation	Normal resistance	Diminished dullness	Atrophy; overlapping lung; pneumo-thorax
		Increased resistance	Cardiac hypertrophy; pericardial effusion
	Normal rhythm	Reduplication . .	Obstruction in systemic circulation; obstruction in pulmonic circulation
		Prolongation of systole	Weakened ventricle; oncoming valve disease
		Accentuation—first sound	Hypertrophy; mitral stenosis
	Normal sounds	Accentuation—second sound	Tension in systemic or in pulmonic circulation
		Irregularity . . .	Fatty degeneration of walls
		Intermission . . .	Embarrassed circulation, or weakness of ventricular systole
		Dropped beats . .	Disordered nervous apparatus
		Murmurs . . .	Disease of valves, of pericardium; abnormal conditions of blood or vessels

TABLE II

Murmurs			
I. Organic			
Endocardial	Friction	Site (a) apex { Left { (b) base { Right { (b) base { Multiple murmurs	Significance Mitral regurgitation " obstruction Aortic obstruction " regurgitation Tricuspid regurgitation " obstruction Pulmonic obstruction " regurgitation Various valvular lesions
Pericardial		(Pericardial . . . Pleuro-pericardial . . .	Pericarditis Pericarditis with pleurisy
Hæmic		At any orifice, but mostly over pulmonic valve	Anæmia, chlorosis, &c.
Venous		In course of large trunk veins	Anæmia, venous obstruction
Arterial		In course of large arteries	Narrowing, or distortion of artery
II. Inorganic			

In the third variety, murmurs of various intensity and character are also sometimes heard in the course of the large arteries springing up from the arch. They may be due to narrowing of the vessel's calibre, or to bendings or displacements of the artery itself. They may also be generated in a roughened aorta, or be caused by dilatations of the arch.

Various murmurs are also frequent in anæmic subjects, being most pronounced at the root of the neck. They consist for the most part of loud boomings, hummings, or even musical sounds ; they often attend both sounds of the heart ; they are generally increased at the end of a deep inspiratory effort, or by pressure of the stethoscope. The most generally accepted theory as to their causation is that known as the 'fluid vein.' In this condition it is supposed that blood flowing through a vessel at one portion of normal calibre enters another widened part of the vessel, but is not sufficient in quantity to fill it, and hence the blood, breaking up into particles or drops, produces a musical vibration.

THE PULSE

Definition.—The pulse is a manifestation of the degree and duration of the excess of blood pressure in an artery which is caused by the ventricular systole. It is not a movement of the vessel, nor is it a measure of the living tension which always exists in an artery. It is, as stated, a measure of the temporary excess of pressure.

The **Normal Pulse** has certain characteristics of frequency, rhythm, force, and tone which it is necessary to fully recognise, so that any alterations in these characteristics may be at once noted as symptomatic of disease.

The *Frequency* of the pulse depends, of course, on the frequency of the heart's action, except in cases of weak ventricular systole, when an impulse may not reach the wrist, or in that rare condition in which two coupled beats of the heart correspond to one beat of the pulse. In the adult the normal pulse-rate is from seventy-two to eighty.

The *Rhythm*, or regularity of force of the pulse, depends on

the regularity of the heart's action ; but occasionally the pulse is irregular in rhythm, although the heart is regular in action, certain of its beats, however, falling short of the radial artery.

The *Force* is due to the amount of vigour of the ventricular contraction, and the amount of blood contained in, and therefore propelled from, the ventricle. The normal amount of blood expelled at each systole is estimated at about six ounces. But it will be easily seen that in cases of valvular incompetence, such as mitral regurgitation, the heart may contract forcibly, and yet the pulse be a feeble one.

The *Character* of the pulse varies according to its tension and the state of the walls of the vessel. Thus, it may come up to the fingers suddenly or gradually ; it may present itself as a long throb, or as a short effort ; and its subsidence may similarly be abrupt or slow.

In every pulse-beat there is a certain amount of dicrotism, but it cannot always be recognised by the fingers. In certain abnormal conditions dicrotism is so pronounced that it can be felt, as well as graphically recorded by the sphygmograph.

In feeling a pulse three fingers should be employed. The first one, nearest the heart, is used to compress the vessel ; the others to estimate the volume of the pulse which is then apparent under them. The artery should also be rolled transversely in its bed, so as to gauge its tension ; and the fingers should also be passed down it longitudinally, in order to detect any irregularities in its coats. Normally, the vessel can only be felt during its pulse-wave, and not during the period corresponding to ventricular diastole. We feel, therefore, a sharp impulse in a low-tensioned vessel, and possibly a second dicrotic wave, also possibly the rebound of the distended artery, and then the tube is lost to the touch till it reappears at the succeeding dilatation. Or in a high-tensioned artery, on the other hand, we perceive a contracted tube, with a heaving impulse, but it can still be felt as a somewhat hardened cord during diastole. We consequently have to estimate the pulse-rate, the blood tension, and the strength and calibre of the vessel. In diseases other than those attended by fever, the rate of the pulse is probably the least important

of its features. Various forms of sphygmograph have been invented to record these different characteristics ; but, after all, the educated fingers are the best instruments, and their cultivated observations constitute the most reliable evidence.

The **Abnormal Pulse** may be excessive in frequency after bodily exertion and mental excitement. It is also increased by food, alcohol, and the upright position. Many diseases also accelerate the pulse-rate, amongst which it is only necessary to mention scarlet and other specific fevers, also exophthalmic goitre, and hysteria. There is also another class of cases (tachycardia) in which the pulse-rate is excessive, amounting to even two hundred beats in the minute, and in which no obvious disease can be detected in heart walls or valves, and in which there is no fever. Bristowe and others have drawn attention to such cases ; and they are generally regarded as due to some functional disorder of the nervous system. In some of the few cases which have come under our immediate care we have been inclined to attribute the cause to disorders of the abdominal viscera and sympathetic system.

An *infrequent* pulse may be said to occur when the rate sinks below sixty in a minute. Forty pulsations in a minute is a rare record, but we have known individuals with a still lower rate.

A *slow* pulse may be quite consistent with perfect health, and, so far as our observations go, it is not an unusual occurrence in tall men, or in those of powerful muscular development. But it also occurs in epilepsy, in jaundice, and in cases of high arterial tension. In recording a slow pulse-rate we must be careful that the apparent slowness is not due to the condition of dropped beat, when only half, or less, of the pulsations reach the wrist. Again, in the condition known as coupled beats, the first may be strong and full and, as it were, obscure the second, which may not be recognised.

As regards rhythm, the pulse may be *intermittent*, or *irregular*, or both. Intermission, again, may occur regularly at, say, the fourth or sixth beats ; or it may occur irregularly, the beat being lost at any period.

Irregularity of rhythm, or inequality of force in different

pulsations, is also subject to variations. The pulse may be rhythmical as regards time, but with beats here and there which vary considerably in force when compared with its general characteristics on this point. Amongst the most frequent causes of this condition are, disordered digestion, the abuse of tea and tobacco, and certain nerve influences which are at present incompletely understood. It is also caused by organic heart disease, notably mitral affections.

Abnormal characteristics due to changes, structural or otherwise, in the artery itself can be, for the most part, included in the two categories, low tension and high tension.

A *low-tensioned* pulse is met with in cases of fatty changes in the heart walls, in fevers, and in obesity, and in some cases of mental anxiety. It is also favoured by general surroundings which increase the bodily warmth.

The *dicrotic* pulse occurs in an exaggeration of low tension, but its features require some notice. Dicrotism is really a secondary wave due to recoil of the dilated aorta. There is always, as stated previously, a certain amount of dicrotism, especially in the carotids, and other arteries near the heart. It is a marked feature in fevers and after hæmorrhages, and is recorded on the sphygmograph by a secondary rise, more or less high, which immediately succeeds the aortic notch, and therefore occurs in the diastolic period. The terms dicrotic, fully dicrotic, and hyper-dicrotic are used to denote the degree of this secondary rise.

The pulse of *high tension*, usually a more serious condition, is due to many different causations. The first which obviously occurs to us is peripheral resistance to the flow of blood. Hence, it may be caused by exposure to cold, by Bright's disease, by gout, and by diabetes; it also occurs in certain cases of anæmia. Other casual factors are, overfeeding and plethora generally; cardiac hypertrophy, whether or not associated with valvular disease; degenerative diseases of the arteries themselves; and irritability of the nervous system.

The following diagrams of sphygmographic tracings will show the points of divergence between the healthy standard and some of the principal abnormal conditions.

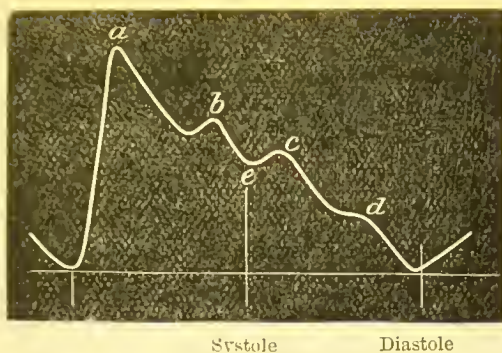


FIG. 16 —HEALTH

a, Percussion wave ; *b*, tidal wave ; *c*, dicrotic wave ; *d*, post-dicrotic wave ;
e, aortic notch.

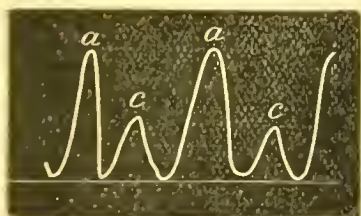


FIG. 17.—DICROTIC PULSE (LOW TENSION)

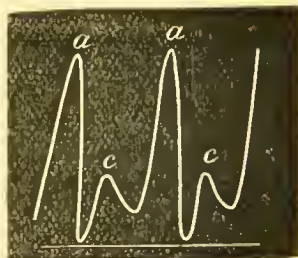


FIG. 18.—HYPER-DICROTIC PULSE

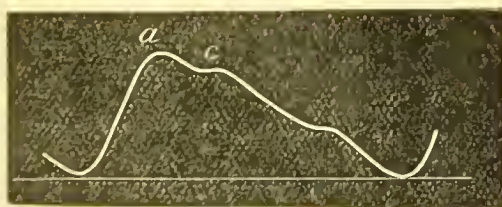


FIG. 19.—HIGH-TENSION PULSE

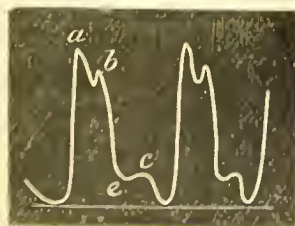


FIG. 20.—COLLAPSING PULSE (AORTIC REGURGITATION)

DISEASES OF THE HEART AND ITS MEMBRANES

PERICARDITIS

Definition.—Inflammation of the pericardium, the fibrous sac containing the heart.

Causation.—It is rarely a primary disease, unless as a direct result of some traumatism. *Fevers.*—Most commonly symptomatic of some specific fever, such as acute rheumatism (about 50 per cent.), scarlet fever, pyæmia, &c. *Inflammation elsewhere.*—It may occur as the result of inflammatory extensions from the pleura, peritoneum, or abdominal viscera; or from intrusion of new growth, such as tubercle, cancer, &c., which act as foreign bodies. The invasion of the sac by aneurysm or by hydatid cyst acts in a similar manner. It is not infrequently a sequel of *chronic Bright's disease*.

Pathology.—There are usually four distinct stages, viz.: hyperæmia, plastic exudation, serous effusion, and adhesion.

In the first stage the appearance of the pericardium is distinctly reddened, the capillaries and smaller blood-vessels being engorged; this is followed by exudation of both red and white blood-cells, proliferation of the epithelium and connective-tissue cells, with exudations of plastic lymph. Thus both layers of pericardium become roughened and thickened. The fibrinous exudation takes various forms and shapes: it may be ribbed, or rolled into tuberculated masses; or it may have a honeycombed appearance, resembling tripe; occasionally the exudation takes the form of thick, shaggy, hair-like processes binding the two layers of pericardium together. In most cases the exudation is most pronounced over the right

ventricle and towards the base. Serous effusion to a greater or less degree accompanies, or immediately follows, this stage. More fluid is probably poured out in weakly subjects. After a time the serous fluid becomes absorbed, and the pericardium, unless the inflammation be very severe, may entirely recover. Generally, however, adhesion, either in patches or by distinct fibrous cords, takes place.

Other secondary pathological conditions to be noted are, the extension of inflammation to the fibrous pericardium and the mediastinal planes of connective tissue; extension to, with fibrinous induration of, the muscular walls of heart; rupture of vessels newly formed in the fibrinous adhesions; pericardial suppuration—which is rare, unless due to pyæmia—and calcareous degeneration of the inflammatory products.

Symptoms vary in gravity according to the intensity of the inflammation. They may be so trivial as to be scarcely recognised; whilst, on the other hand, they may constitute a condition of the utmost peril. The general symptoms are fever (temperature 100° to 105°), with vomiting, thirst, marked dyspnœa, lividity, and a pinched and anxious expression. The pulse is rapid, thin, and occasionally jerky or intermittent. The severer cases are often attended with wild delirium, followed by convulsions and coma.

The local signs are tenderness over the precordial region, with pain on movement or with deep inspiration. Auscultation in the early stage reveals a rough 'to-and-fro,' or 'frou-frou,' friction sound, apparently quite superficial, and best heard, as a rule, down the middle of the sternum and over the right side of the heart. Occasionally a pericardial rub can be distinctly felt.

If the inflammation subsides, the above physical signs become less pronounced in a few days, and finally disappear, the pericardium regaining its normal state, or its cavity becoming obliterated by adhesions. Usually, however, more or less serous effusion occurs. Then the area of heart's dulness becomes increased; it assumes a marked pyriform shape, with the base downwards and outwards; and the apex-beat is either entirely lost, or diffused, or slightly tilted upwards.

The lateral and the upward extensions of dulness vary according to the amount of effusion, and, as a rule, so does the degree of percussion resistance. Should effusion become excessive, signs of pressure on neighbouring organs and vessels become evident. For example, the patient may have hiccough, dysphagia, engorgement of the veins of head and neck and upper extremities, together with œdema of intercostal spaces, and displacement of lungs and of abdominal viscera. The temperature of the lower extremities is lower than that of the upper, owing to pressure of the pericardial effusion on the aorta. In children, pallor is often a marked symptom in pericardial effusion.

TERMINATION OF THE CASE.—(1) The patient may entirely recover, and the pericardium resume its normal state. (2) He may die, during the height of the attack, from fever, coma, or syncope. (3) The inflammation may end with suppuration, and death ensue after a prolonged illness. (4) Adhesions may form to a greater or less extent, to be followed by myocarditis, dilatation of ventricles, and incompetence of valves, with their usual sequels, anasarca and albuminuria.

Diagnosis.—The friction sounds may resemble those produced by obstructive and regurgitant disease of the aortic valve. But in pericarditis the double sound is more superficial and rougher in character; it is usually increased by pressure of the stethoscope. Further, the rough murmur is not limited to the aortic area, and it may vary in intensity and in situation from day to day.

Prognosis.—The disease is not fatal, as a rule, except there have been one or more previous attacks, when adhesions tend to embarrass the heart's action. The pyæmic form is obviously a serious condition.

Treatment.—Apart from the treatment of acute rheumatism, scarlet fever, or other acute disease of which pericarditis may be an accompaniment, we must also attack the seat of inflammation itself. For this reason it were perhaps as well to look on pericarditis as of the same nature as an inflamed joint, and treat it accordingly. Therefore, absolute rest in bed is essential: The patient should not be allowed to rise in his bed.

The diet should consist of light liquid nourishment. In strong patients, with a severe attack, we may apply six to twelve leeches over the precordial area. Others may receive benefit from the application of linimentum cantharidis over the same region ; then, on the following day, aspirate the blister with a hypodermic syringe, subsequently injecting into it about five minims of the hypodermic solution of the acetate of morphia. We thus relieve pain and give sleep by the one and the same means. This treatment may be repeated every two or three days. The cardiac region should also be protected by cotton-wool. Alcoholic stimulants may be requisite from the beginning, but generally it is best to withhold them until absolutely required. Symptoms which demand alcohol are, pallor and cold skin, a feeble first sound of the heart, or an irregular feeble pulse. If the effusion be excessive, the question of aspiration of the pericardium must be considered ; but it should be performed only as the last resource, and not at all if other symptoms point to impending death. After the acute stage has passed off, if the effusion shows little sign of absorption, we may hasten this by iodine or other stimulating liniment over the heart's region ; whilst internally we administer small doses of iodide of potassium, nitrate of potash, together with mild aperients. The patient, however, should not be violently purged, and the use of a bed-pan must be enforced. Beyond this we must improve his general condition by tonics—iron and quinine, separately or combined.

Prolonged rest, with freedom from anxiety and exertion till long after all inflammatory signs have disappeared, are absolutely imperative.

ENDOCARDITIS

Definition.—Inflammation of the endocardium or internal lining membrane of the heart. The disease may be acute or chronic.

Causation.—Rarely a primary disease. *Age and Sex.*—Men are more subject than women, and it occurs oftenest in early adult life. *Ferers.*—The majority of cases are secondary to

acute rheumatism, scarlet fever, measles, pyæmia, or other specific fever.

It also frequently appears as a sequel of chorea (possibly another form of rheumatism), and of Bright's disease. Exposure to hard life, with insufficient food, alcoholism, and all conditions which tend to a damaged system, are pre-disposing causes.

Pathology.—The disease may be congenital, when the right side of the heart is most affected ; otherwise, in a large preponderance of cases, it involves the left heart only (90 per cent.). The inflammatory course runs through stages similar to those found in inflammation of other membranes. There is first engorgement of the capillaries and smaller vessels ; this is followed by cellular proliferation, with exudation of lymph on the surface of the endocardium, and consequent opacity thereof, thickening of chordæ tendineæ, and swelling of the muscoli papillares.

Similar phenomena take place in the interior, and on the surface of, the valves. The mitral valve is usually the first to be attacked, and especially that right curtain which is continuous with the left posterior cusp of the aortic valve. The effect of the inflammatory process on the valves is to cause thickening and contraction of the curtains, together with the deposit, along their free edges and surfaces, of granulations or vegetations of different forms and shapes, with consequent interference with their mechanism. The incompetence of these valves varies according to the amount and extent of these warty excrescences and the degree of contraction of the flaps of the valves themselves. In the aortic valve it is the ventricular surface which is most affected, whilst in the mitral valve the inflammatory deposit is most marked on its auricular aspect, and it may even extend on to the walls of the left auricle. Nor are the pathological changes limited to these structures, but they may even invade the fibrous rings and septa which separate auricle from ventricle, and the ventricles from each other ; and may also extend to the aortic endothelium.

The granulations just described may remain quiescent and

unaltered throughout the patient's life ; but often they undergo degenerative changes, which cause them to become brittle and detached as emboli, which are thus swept into the circulation until they are arrested by some artery of too small a calibre to allow them to pass.

The surfaces from which they are detached are thus converted into minute ulcers, which may heal ; or the destructive process continues till perforation of the curtain and destruction of the valve take place (see Ulcerative Endocarditis). Further, the changes are not limited to the valves. The endocardium lining the heart cavities may undergo similar inflammatory changes which extend to the heart muscle causing softening of heart walls and bulgings (ventricular aneurysm) ; but in all cases such inflammatory process is patchy and limited, not general. Destructive ulceration and rupture of chordæ tendineæ occasionally take place.

Symptoms.—The general symptoms are not pronounced. There is usually slight fever, as indicated by some elevation of temperature, which is attended by thirst and general malaise, also cardiac discomfort with palpitation and quickened pulse. The best evidence, however, is obtained by physical signs. The existence of muffled heart-sounds, or of a prolonged systole, and finally of a murmur in a patient suffering from acute rheumatism, chorea, or other disease prone to be complicated by endocarditis, is practically enough evidence on which to found a diagnosis. We must be careful, however, not to confound a recent endocardial murmur with that of pericarditis or of old valvular disease. In the former case there is sooner or later an increase of pericardial dulness ; we infer that the latter condition exists by finding marked signs of cardiac hypertrophy.

Diagnosis.—(1) FROM CHRONIC VALVULAR DISEASE.—The murmurs in old endocarditis are rough and harsh ; and there are almost invariably signs of cardiac hypertrophy.

(2) A HÆMIC MURMUR would be diagnosed by its occurrence, for the most part, in a chlorotic girl, or an anæmic subject, without any history of previous acute rheumatism, and without cardiac hypertrophy. The murmur is also soft and

blowing in character, and most frequently heard over the pulmonic valve during systole.

Treatment.—The general treatment consists of a continuation of the same measures which we are using to combat the disease (say acute rheumatism, diphtheria) of which endocarditis may be a complication. Beyond this, absolute rest is necessary. We may thus prescribe opium to procure sleep, and digitalis to steady the heart's action. Iodides have been recommended with a view to their absorbent action. The diet should be light and not stimulating; yet alcohol may be needed if danger threatens from syncope. The bowels should be relieved regularly by laxatives or enemata.

During convalescence iron tonics are indicated. Perhaps the most essential treatment is prolonged rest in bed (quite six weeks) after all signs of endocarditis have disappeared; since the disease may remain and lurk behind, although auscultation gives negative evidence. It is no uncommon thing for a patient to return, six months after supposed cure, with marked symptoms of valvular lesion, although we had previously deluded ourselves that the valves were intact.

ULCERATIVE ENDOCARDITIS (MALIGNANT OR SEPTIC ENDOCARDITIS)

Definition.—An ulceration of the endocardium, due to septic poisoning, mostly affecting the valves of the left heart; which is followed by the detachment of numerous emboli, producing septic infarcts in different organs.

Causation.—(a) **PREDISPOSING.**—*Age.*—It especially occurs in young adults. A *chronic endocarditis* may suddenly take on septic conditions. It may be secondary to some *acute febrile disease*, such as pneumonia, acute rheumatism, scarlet fever, erysipelas.

(b) **EXCITING.**—Some specific organism, which does not as yet appear to have been sufficiently determined. Streptococci and staphylococci have been discovered in the minute ulcerations, as well as the specific organisms of pyæmia and of other pre-existing fevers.

Pathology.—The disease may begin, though rarely, as an initial lesion ; but more frequently it is grafted on a chronic endocarditis. The most usual seat of infection is a valve, especially the mitral ; but any valve may be involved. The disease may extend from the mitral flaps backwards to the lower part of the left auricle, or from the aortic cusps to the aortic arch, causing a specific endarteritis. The lesions are either in the form of new growths or vegetations, or of ulcerative destruction of surface with suppuration. Vegetations are small exerescences, of greyish or greenish tint, and often coated with lime salts. They consist essentially of fibrin, with granulation tissue at their bases, the neighbouring cellular tissues being proliferated as the results of inflammation.

The ulcerations may be superficial, and involve only the endocardial layer, leading thus to the formation of aneurysm of the valve ; or they may be deep, and perforate, or even entirely destroy, the valve curtains or their chordæ tendineæ. The bases of the vegetations frequently contain minute abscesses, which may extend to the myocardium. When the affection is mural, vegetations are the most marked products ; still even here there may occur perforation of the heart's walls or of its septa.

Subsequently infective emboli are detached from these vegetative surfaces, and are carried by the blood-current to different organs : they lodge especially in the kidneys, the spleen, the brain, and the intestinal vessels, where they produce inflammatory infarcts and consequent suppuration. These infarcts are, in fact, reinfections at these organs ; but the emboli produce, in addition, all the phenomena of obstruction to the vessels and of hæmorrhages which are common to non-malignant emboli.

Symptoms resemble those of pyæmia, together with those of endocarditis. There is high fever, usually of the irregular or intermittent type ; but there may be two or more exacerbations and remissions in the day ; the temperature curve is consequently very 'zigzag' in character. The fever is accompanied by rigors, sweating, diarrhœa, an enlarged spleen, painful distension of the belly, with a dry tongue, and

other typhoid symptoms. There is often, besides, a petechial eruption on the skin, and also a tendency to drowsiness or delirium and coma, with other signs of meningitis. Suppurative inflammation of joints is also a common complication. If a cardiac murmur had existed prior to the onset of malignant endocarditis, it becomes louder and harsher ; but it is not necessary that a murmur should even exist. The patient, however, generally complains of cardiac pain and oppression, with dyspnœa. The duration of the disease may vary from a few weeks, in a primary attack, to some ten or twelve months when engrafted on old valvular affection.

Diagnosis.—Often extremely difficult. (1) It resembles TYPHOID FEVER in many respects, but ulcerative endocarditis lasts longer, as a rule ; the exacerbations and remissions of temperature are more marked, and occur two or three times a day ; the rash also is petechial.

(2) FROM AGUE.—By the absence of malarial infection ; by the splenic enlargement not being so pronounced ; and by the non-efficacy of quinine.

(3) FROM PYÆMIA.—The probability is that malignant endocarditis is a form of pyæmia, and that the joints become involved, and abscesses form secondarily to the suppurating endocardium, instead of the infection starting as usual from some superficial suppurating surface.

Prognosis.—Always bad. Occasionally cases recover when recognised in the initial stages and treated by prolonged rest.

Treatment.—Little can be done. The body must be kept at perfect rest ; movement caused by the use of bed-pan even is attended by danger. We may try the effects of large doses of bromides with salicylates, or treat the pyrexia with heroic doses of quinine (grs. xv. t.i.), as in septicæmia. Dr. Sansom has recommended the use of sulphocarbolates. Check diarrhœa by the usual astringent remedies. Opium is the most reliable drug.

VALVULAR DISEASE

Definition.—Conditions, either congenital or acquired, by which one or more of the cardiac valves are rendered

mechanically imperfect. The various changes may be limited to the rings of the valvular orifices, or to the flaps, or may involve both.

Causation.—(1) ACUTE ENDOCARDITIS, secondary to acute rheumatism, pyæmia, and other specific fevers, is the most common cause, especially in early life. (2) DEGENERATIVE CHANGES, commoner in advanced life, such as atheroma, syphilitic lesions, which cause adhesions of valve flaps, &c. (3) DILATATION and INSUFFICIENCY of orifices without actual disease of valve curtains. (4) VARIOUS MALFORMATIONS (developmental). (5) ANEURYSM OF VALVE, often secondary to ulceration. (6) RUPTURE OF CHORDÆ TENDINEÆ, of flap or of other part of valvular apparatus, usually caused by some severe strain or exertion, supervening generally on a previously diseased valve.

In ninety per cent. of cases the lesion occurs in the left side of the heart. Statistics vary as to frequency of these. Our experience of cases of all ages shows that the frequency of the various lesions is according to the following order, viz. : mitral regurgitation, aortic obstruction, aortic regurgitation, mitral obstruction. Aortic disease, however, is especially a disease of advanced life, whilst mitral disease is more frequent in the young. As regards *sex*, males are more prone than females, on account of their greater exertions and exposure. Mitral stenosis, however, is perhaps more frequent in females, and is said to be associated with uterine disease.

AORTIC VALVE DISEASE

Aortic Obstruction (STENOSIS)

Causation.—It may be due to (1) roughening and narrowing of the *aorta* immediately above the valve ; (2) to granulations, thickenings, or adhesions of the cusps ; or (3) to narrowing of the aortic ring itself (see fig. 21).

These conditions give rise to a systolic murmur heard at the base over the aortic orifice. (See Murmurs, p. 400.)

Occasionally the murmur is best heard over the manubrium sterni, and occupies the end of systole (post-systolic). It is

then generally due to the roughening of the aorta, the valve itself possibly being healthy. In all cases of aortic obstruction there is probably some slight regurgitation in addition, although the conditions may not be such as to generate

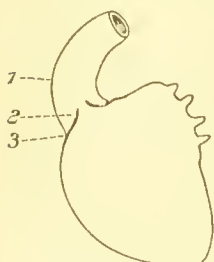


FIG. 21

a reflux murmur. The systole also is often attended by a thrill felt over the base. The changes in the heart which ensue are : (1) hypertrophy of the left ventricle, the amount varying according to the extent and the duration of the obstruction ; (2) dilatation of the left ventricle, this being due, possibly, to the slight regurgitation, but more probably as a result of the stretching of the muscular walls.

In marked stenosis the pulse is slow, thin, and prolonged at the wrist, the vessels being only imperfectly or gradually filled. When the obstruction is very pronounced, a safety overflow through the mitral orifice may occur, owing to dilatation of the ventricular cavity causing insufficiency of the mitral valve.

Symptoms are not so distressing as in other forms of valve disease. In advanced cases, however, marked dyspnoea with occasional syncope may occur.

AORTIC REGURGITATION

Causation.—(1) DILATATION of aorta above, or of ventricle beneath, the valve. (2) MORBID CHANGES in the valve cusps, *e.g.* : senile degenerations, ruptures, perforations, vegetations, inflammatory thickenings, and adhesions, either to neighbouring cusp or to the edges of the sinus of Valsalva.

Signs and Symptoms.—The cardiac area is increased and often bulged owing to early dilatation of the ventricle ; the impulse is outside and much below the nipple ; a diastolic thrill may at times be felt ; there is marked jerking pulsation of the carotids and larger vessels. Pulsation may be detected in the capillary vessels of the retina, of the lips and skin, and of the matrix of a finger nail. A murmur of long duration, generally filling the diastolic period, is heard with greatest

intensity over the aortic valve, or end of the sternum, or occasionally at the impulse (see Murmurs, p. 400). Although the amount of regurgitation is roughly indicated by the duration of the diastolic murmur, a second sound heard in the carotids at the root of the neck would show that the valve is not entirely incompetent.

The pulse is characteristic: the wave of blood filling an artery of low tension comes up readily and suddenly to the finger with some force; but the wave is not sustained, and the pulse collapses immediately, owing to reflux of blood through the aortic semilunar valves. This condition is better observed when the patient's arm is held vertically upwards; it is known as the collapsible or 'water-hammer' pulse, or the pulse of unfilled arteries, and is called after Corrigan, who first drew attention to it. The arteries are tortuous, elongated, and mobile with each systole, and their calibre enlarged generally. It is rare to find aortic regurgitation pure and simple; it is usually accompanied by more or less obstruction. It is also attended by palpitation, pallor, and dyspnoea; but the degree of dyspnoea and other distress varies according to the amount of regurgitation and the amount of compensatory hypertrophy. If the ventricle be sufficiently dilated and hypertrophied so as to allow of an amount of blood to be propelled into the arteries which is enough, allowing for regurgitation, to keep up a fair tension, then dyspnoea is not urgent. Subsequently, should compensation fail, there are signs of excessive dilatation, without corresponding hypertrophy. Dilatation is followed by mitral insufficiency with its attendant conditions. The failure of compensatory hypertrophy is by some thought to be due to impaired nutrition of ventricle, owing to imperfect filling of the coronary arteries.

Cases of aortic insufficiency are liable to alarming syncope, with or without angina, which may terminate fatally. Dyspeptic symptoms also are common: flatulence, sickness, and distress after food being frequently urgent, and difficult to relieve; they always increase the gravity of the case. Nightmare and disturbed sleep are complained of; the patient also has a characteristic pallor.

MITRAL REGURGITATION

Causation.—(1) THICKENING OF VALVE. (2) ADHESIONS OF CURTAIN to neighbour, or to ventricle wall. (3) VEGETATIONS. (4) DEGENERATIVE CHANGES (calcareous). (5) RUPTURE OF CURTAIN. (6) SHORTENED OR RUPTURED CHORDÆ TENDINEÆ. (7) DILATATION OF LEFT VENTRICLE (secondary to aortic disease, or due to fatty changes in muscular walls). (8) DILATATION OF AURICLE (in divers).

Signs and Symptoms.—The cardiac dulness is enlarged, especially transversely ; there is an increased impulse, with, in advanced cases, a bulging of the precordial area. A murmur is heard at the apex-beat, generally loud and blowing, and usually of the same character throughout, but varying in intensity in different cases. It occupies the period of the first sound, which it replaces or, at least, obscures. If the first sound is heard through the murmur, the valve is probably rigid and stiff only. The murmur is propagated in the direction of the reflux blood-current, hence it can be detected round the left margin of the heart, towards and in the left axilla, and posteriorly at the apex of the left scapula.¹

In addition, the period of systole is prolonged, and the second sound over the pulmonic artery is accentuated and reduplicated. Occasionally a murmur may be heard at the apex, and there only ; this is perhaps due to some local roughening of the ventricle's inner surface.

We estimate the amount of the valvular insufficiency by the extent of the hypertrophy ; and, as a rule, the greater the force of the regurgitant stream, the louder the murmur. Consequently, when compensation fails, a murmur may become weak or almost inaudible. The pulse is small, quick, of low tension, and regular, except when compensatory hypertrophy fails ; then it gets irregular and intermittent. Irregularity, however, may be an early sign, inasmuch as it is

¹ We think that undue stress is laid on this point as being diagnostic. We find that it is merely a matter of intensity of noise. Some weak mitral regurgitant murmurs are not to be detected at the angle of the scapula, whilst some loud aortic murmurs may be.

in great measure due to the varying tension of the circulation during inspiration and expiration. Thus more blood is expelled into the aorta when the tension in the left auricle and capillaries of the lungs is greatest. There is usually a well-marked circumscribed blush on the cheeks, together with blue lips and other signs of circulatory impediment.

Dyspnœa and palpitation, though often present, are not necessarily symptoms, at least in the early stages when compensation is maintained; indeed, mitral insufficiency may exist for years with little or no distress. So long as hypertrophy of the left ventricle is maintained to a proper extent, all is well; but when it fails, the left ventricle dilates, the left side of the heart and the pulmonary vessels become engorged, and the signs of backward pressure of the blood are manifest. So that before œdema supervenes, dyspnœa has invariably existed for some lengthened period. In addition, when the disease is advanced the patient often complains, as in aortic insufficiency, of indigestion accompanied by enormous accumulations of wind in the stomach; he has a grasping or 'clutching' pain over the cardiac region, and he frequently dreads the night on account of his disturbed sleep and distressful dreams.

MITRAL OBSTRUCTION (STENOSIS)

Causation.—(1) CONSTRICTION OF THE AURICULO-VENTRICULAR RING. (2) ADHESION OF SEGMENTS OF THE VALVE, causing a flattened or funnel-shaped communication between the two cavities, with a button-hole aperture below. (3) AGE.—Most common in early adolescence, as it is nearly always secondary to acute rheumatism. The inflammatory affection is not limited to the valve curtains, but, extending to the auriculo-ventricular ring, apparently gives rise to narrowing and arrest of development of the aperture. Regurgitation is frequently a concomitant condition.

Signs and Symptoms.—The cardiac dulness is increased transversely to the left, owing to hypertrophy and dilatation of the left auricle and hypertrophy of the right ventricle. The left ventricle is normal in size, or, if altered, it is shrunken

and atrophied. A perceptible thrill or tremor is usually easily felt during diastole at the apex. There is an increase of pulsation in the epigastrium consequent on hypertrophy of the right ventricle. On auscultation a murmur or murmurs occupy some portion or the whole of the diastolic period. The thrill attended by murmurs is usually observed in the early stages. If the second sound caused by the closure of the aortic valve is well heard, it is a good sign. It shows that the ventricle is fairly well filled. In the later stages both thrill and second sound disappear, owing to the current of blood passing through the ventricle being insufficient to generate a thrill, or to fill the aorta and throw down the semilunar cusps. (See Murmurs, Mitral Obstruction, p. 399.)

Soon after stenosis has become well-marked there are signs of congestion of the bronchial tubes and lung parenchyma. Thus, dyspnœa and hacking cough are early symptoms; there is profuse mucous expectoration, often tinged with blood; or there may be free hæmoptysis, which often affords relief. The congestion of the lungs, however, is permanent, causing 'brown induration,' and is often attended by pulmonary apoplexy. The pulse is feeble, but regular, until the advanced stage, when intermission occurs, this being probably due to some beats not reaching the periphery. The subsequent clinical conditions are much the same as in mitral obstruction.

PULMONIC OBSTRUCTION (STENOSIS)

Causation.—Usually due to some congenital defect, or to aneurysm of pulmonary artery or its branches. A murmur resulting from organic disease, except it be ulcerative endocarditis, is rare.

Signs and Symptoms.—The cardiac area is increased in the direction of the right ventricle. Epigastric pulsation, together with sighing, dyspnœa, and cyanosis, due to deficient aëration of blood, are usually noted. A systolic murmur is heard over the pulmonic valve on the left side of the sternum. (See Murmurs, Pulmonic Obstruction, p. 401.)

The condition is very rare.

PULMONIC REGURGITATION

Causation.—Some congenital defect usually. Cases have been known to occur from rupture of cusps in divers and others with impeded pulmonary circulation.

Signs and Symptoms.—Much the same as in pulmonic obstruction. For recognition of Murmurs see p. 401.

TRICUSPID REGURGITATION

Causation.—Actual disease of the valve curtains is very rare. The condition is usually secondary to chronic disease of the lungs or of the left side of the heart; there is thus an accentuation of the normal 'safety-valve' overflow of the tricuspid valve. This may be aggravated by the dilatation of the right ventricle, with consequent comparative shortening of the musculi papillares and increase in the calibre of the auriculo-ventricular ring; the valve segments thus fail to meet.

Signs and Symptoms.—The cardiac dulness is increased transversely, and upwards to the right, owing to hypertrophy of the right ventricle and dilatation of the right auricle. Epigastric pulsation is well-marked. In advanced cases pulsation, which may come and go, may be seen in the external jugular and other veins of the neck, the valves being overcome. Pulsation has also been described as occurring in the liver, but we have never yet been satisfied that it is true pulsation apart from 'jog' from the enlarged right ventricle. Venous engorgement of the liver, kidneys, and lower extremities comes on early, with all the symptoms of backward pressure of the blood.

On auscultation a systolic murmur is heard over the lower end of the sternum, at about the level of the fifth cartilage; it is limited in area, of a soft (seldom rough) blowing character.

The disease occurs much more frequently in women.

Occasionally a systolic murmur may be heard loudest over the middle of the right ventricle, owing to some thickening of the endocardial surface of the ventricle, or to roughening of the ventricular aspect of the tricuspid valve, but without regurgitation.

TRICUSPID OBSTRUCTION (STENOSIS)

Causation.—Nearly always congenital, and associated with malformation. Extremely rare.

Signs and Symptoms would be those of obstruction to the venous current, viz. hypertrophied and dilated right auricle, early œdema of legs, and congestions of abdominal viscera. Life is seldom prolonged.

Prognosis of Valve Diseases.—Valve disease is always serious ; and in any form, with perhaps the exception of aortic obstruction, the duration of life is shortened and always uncertain. The following points will, however, influence us in our prognosis :

(1) **CAUSE.**—Valve disease caused by degenerative changes is more serious than when secondary to acute rheumatism.

(2) **VALVE AFFECTED.**—The order of gravity in valvular diseases is as follows : aortic regurgitation is the most serious, mitral obstruction is more serious than mitral regurgitation, whilst aortic obstruction is least serious of all. Some authorities account mitral obstruction as being the most unfavourable.

(3) **EXTENT AND CONDITION OF LESION.**—It naturally follows that the more the obstruction, or the greater the leakage, the more urgent the danger. Nevertheless, aortic regurgitation appears to be a more favourable disease when complicated by mitral regurgitation, the latter condition acting as a safety-valve overflow to a distressed ventricle. Certainly dyspnoea is less urgent : there is less diastolic shock, the bursting precordial sensations and anginoid attacks are less pronounced, and sufferers appear to get through a certain amount of work with more comfort than when the mitral valve holds. Again, the presence of numerous vegetations on a valve makes the disease more formidable, from the liability to embolism.

(4) **OCCUPATION.**—Obviously those who have to labour hard for a livelihood are in a worse position than those who can afford to live comfortably at ease. Frequently one meets with a hospital case and a private patient, both with apparently

identical lesions, and yet the private patient long survives his fellow-sufferer.

(5) COMPLICATIONS.—The gravity of valve disease is increased in all diseases and conditions which produce degenerative tissue changes—such as syphilis, Bright's disease, fevers, &c.

Treatment.—(1) PROPHYLACTIC.—A recurrence of endocarditis should be strictly guarded against; a life which may not be very seriously shortened by one attack is made much more precarious by subsequent seizures. Therefore the patient should avoid cold, damp, unnecessary exposure, exertion and excitement, and all excesses of diet or of alcoholic liquors, which tend to produce endocardial inflammation.

(2) PALLIATIVE.—There are certain general rules of treatment which apply to all forms of valvular disease. We cannot alter the injury to a valve; it is probably never cured, except perhaps by a prolonged rest in a recent affection; and the tendency of the lesion is towards aggravation rather than amelioration. We must, therefore, promote or control the various secondary disorders and conditions imposed by Nature. Hypertrophy of the muscular walls and dilatation of cavities are the chief effects of the disease on the heart itself. Both, therefore, are advantageous to a certain extent, being Nature's efforts to repair; but both may be excessive, and therefore a source of danger. It is the maintenance of a happy medium between deficiency and excess which it should be our aim to establish. This medium is called 'compensation.'

Treatment is markedly beneficial in early cases, whilst the muscular tone in the vessels and the condition of the tissues are not irretrievably damaged.

Diet should be nutritious, digestible, and enough to maintain tissue waste, but not too abundant. Excess of food is wrong. As a rule, too much is given, and often of indigestible character. We find it a good rule to diet a patient much as if he were an athlete preparing for a contest. He should also abstain from fluids as much as possible, as they increase the blood pressure. A somewhat dry

diet is therefore advisable, and is soon readily tolerated, from the relief which it brings. Tobacco and alcoholic stimulants are injurious, except in moderation and under skilled supervision. The bowels should have daily and easy relief. Examination of the urine should be made from time to time, as its quantity and specific gravity are guides to the amount of the vascular tension. In all heart troubles a good rule is to treat the dyspeptic symptoms first.

The digestion should be regulated by tonics, such as quinine, nux vomica with alkalies before meals, or by mineral acids after food. Maintain the patient's muscular power by moderate exercise on the flat, and allow him, if possible, cheerful occupation and pursuits. The idea that a sufferer from cardiac disease is doomed to a life of complete rest and inactivity is an erroneous one; a little muscular exercise and mental excitement, short of undue exertion or of excessive emotion, are of distinct advantage. Pregnancies should be avoided. Excessive sexual excitement is also injurious to both sexes.

No cardiac stimulants should be given until compensation fails; it is a flagging horse which requires the spur, and much harm can be done by the indiscriminate prescribing of digitalis and other remedies before they are wanted. A diseased heart responds so much more readily to cardiac tonics when they are withheld until needed. Failure of compensation, and the consequent demand for digitalis, will be evinced by dyspnoea, dropsy, and anæmia. We then direct our treatment to the disorders of the various systems which are the sequelæ of valvular disease ('failing compensation'). The following is a short description of these sequelæ.

(a) *Respiratory System*.—Pulmonic complications ensue earliest, and are the most serious. Congestion always yields in the early stages to prolonged rest in bed, supplemented by mild purgation, or, if necessary, bleeding by leeches over the bases of lungs. In advanced cases complicated by bronchitis, pleural effusion, œdema of lung, and perhaps by a low pneumonia, further remedies are needed. They are all merely local manifestations of circulatory stasis and œdema. They

may be considerably diminished by lessening the quantity of fluids consumed, and by restoring the balance of circulation by one or other of the cardiac stimulants combined with iron,¹ or by soothing expectorants with iodide of potassium.²

(b) *Digestive System*.—Both lobes of the liver being enlarged and painful from engorgement, we can obtain relief best by saline purges and other depletory measures. As a rule, the congestion soon yields to such treatment. If the liver be very painful, and the circulation much embarrassed, nothing succeeds so quickly as bleeding. Six leeches over the right hypochondrium will often work wonders. Check vomiting, diarrhoea, and hæmorrhages, if severe; mild attacks, however, may do good, as the intestinal canal, being engorged, is in an unfavourable condition to absorb food and medicines. We thus afford relief by gentle purgation with mercury, podophyllin, colocynth, rhubarb, aloes. In advanced cases more drastic purgatives may be prescribed, such as croton oil, elaterium, gamboge; but they should be given with care, and their effects watched. Elaterium combined with digitalis is especially useful in the ascites of old-standing disease.

(c) *Circulatory System*.—Anasarca is best treated with squills, digitalis, scoparium, acetate of potash, and other diuretics.³ We may puncture the limbs, when greatly œdematous, or insert Southey's trocars; but all wounds of the surface should be protected by strict antisepsis, and, if possible, avoided. The diet should be 'dry,' with as little fluid as can be tolerated. Dyspnœa, often most distressing at night, may be relieved by mustard and linseed poultices, or by rubefacient liniments applied over the front of the chest, so as to draw blood to the surface.

(d) *Nervous System*.—For insomnia few remedies succeed so well as morphia (gr. $\frac{1}{2}$), either by the mouth or subcutaneously.

¹ R̄. Tinct: Ferri Perchlor: m̄x.; Tinct: Digitalis m̄vij.; Spt: Chloroformi m̄x.; Aquam ad ʒj. Misce.

² R̄. Ammon: Carb: gr. iv.; Tinct: Scillæ m̄xv.; Potass: Iodidi gr. v.; Aquam ad ʒj. Misce.

³ R̄. Tinct: Digitalis m̄x.; Tinct: Scillæ m̄xx.; Potass: Acetatis gr. x.; Decoct: Scoparii ad ʒj. Misce.

It is less dangerous than chloral, and is not contra-indicated by even a medium degree of albuminuria. Bromides combined with hyoscyamus are also valuable. Sulphonal may also be tried, but its efficacy is temporary only. Urethane is the best and safest hypnotic ; it has no taste, and may be given in ten- to twenty-grain doses, when it produces most refreshing sleep. Paraldehyde is also an excellent and safe hypnotic ; it gives sleep without depressing the heart's action like chloral hydrate does. It should be given in half-drachm doses in chloroform water every hour until sleep ensues. Amyl nitrite is employed in the anginoid attacks of aortic regurgitant, and in mitral obstructive disease, on account of its efficacy in lowering blood pressure.

It will also be found that the position of the patient may be changed from time to time with advantage. A cardiac sufferer often sleeps comparatively comfortably in an arm-chair with his head resting forward on the back of another chair.

Cardiac Tonics.—There are many drugs which act as direct cardio-vascular tonics ; we will briefly consider the most important. As a cardiac stimulant digitalis is best by far. It is, in a large series of cases, worth more than any other combination of drugs that we know of. We revert to it after other medicines have been tried, and failed. Still, there are certain cases in which, from some idiosyncrasy, digitalis does not succeed so well as other remedies. The tincture has the best effects on a flagging heart ; the fresh infusion appears to act quickest as a diuretic, as its speedy, diffused action improves the blood pressure in the renal arteries and Malpighian tufts, and thus favours diuresis. Digitalis acts by restoring the heart's rhythm and strengthening its beats ; it is especially valuable in mitral stenosis and the consequent venous engorgement, as it prolongs the ventricular diastole, and so gives a labouring left auricle more time in which to send on its contents. In some cases, however, it may cause severe nausea, sickness, palpitation, and prostration. In administering the drug note the daily quantity of urine, as it should be discontinued if a diminishing amount is excreted ; nor should it be given when arterial tension is high, or in cases of extreme

dilatation of the left ventricle. We are not in favour of digitalin ; a given dose is not so efficacious as the corresponding dose of digitalis, whether in form of tincture or infusion. There appear to be other principles in the crude drug which are useful, besides digitalin.

Strophanthus is another valuable and safe remedy. It is a cardiac tonic, a diuretic, and quickly relieves precordial pain and dyspnœa. The tincture is the best preparation. It should be given in small but increasing doses (ʒij.), combined with stimulants and antispasmodics.¹ It is most useful in aortic regurgitant disease, as its power of lengthening diastole is not so great as digitalis, nor has it so marked a cumulative action.

Convallaria majalis is a simple cardiac tonic and quite a safe remedy. In its action it causes slowing, and increases the force of the heart-beats.

It will be frequently found in failing compensation that the above drugs individually disappoint us after a time, but that a combination of all three has often an effect little short of marvellous.

Belladonna is of great service in cases where digitalis has disagreed. It lessens palpitation, diminishes the sensitiveness of the endocardium, and lessens irritability of the stomach. As a local application (plaister) it sensibly diminishes true cardialgia.

Caffein is a valuable diuretic. It may be prescribed as the citrate (gr. iij.), in combination with the tinct. convallariæ majalis (ʒx.).

When drugs fail to restore compensation we may resort to blood-letting. The pulse often improves after bleeding, as the circulation then becomes less embarrassed. Leeches may be applied over the sternum, or over the bases of lungs, or we may 'wet' cup. A series of blisters followed by poultices also help to restore the circulatory balance. Bleeding, however, is contra-indicated in debilitated subjects with advanced disease, or when there is pneumonia.

In all forms of valvular disease we should bear in mind

¹ R. Tinct. Strophanthi ʒiv. ; Spt : Aminon : Aromat : ʒxx. ; Spt : Ether : Co : ʒxx. ; Aquæ Chloroformi ad ʒj. Misce.

that the greater part of the blood is behind the diseased valve, therefore in the venous system, and that the arterial system is comparatively empty. We therefore assist those drugs which are direct cardiac stimulants by other measures, such as diaphoresis (hot baths, liq. ammon. acetatis) and diuretics. But in the treatment of the various complicated conditions occurring in advanced valvular disease we must be guided principally by those symptoms which appear at the time to be of most preponderating importance.

The treatment of aortic regurgitation requires a few special points. We find it advisable to forbid the use of digitalis. In this form of disease a sudden failure of the left ventricle is the chief danger. Digitalis, by causing a more vigorous ventricular systole, allows a greater volume of blood to be expelled at each beat ; but the drug also lengthens the diastolic period, and so allows a greater regurgitant pressure in an already dilated ventricle, which may suddenly give up the struggle. Still, some cardiac tonic is necessary, and we prefer strophanthus in combination with ether. But authorities are by no means agreed on this subject. We may also, from time to time, prescribe strychnia, iron, quinine ; or send the patient to a bracing climate, if possible.

MYOCARDITIS

Definition.—Inflammation of the heart muscle.

Causation.—Rarely a primary affection. It is almost invariably a sequel to endocarditis or pericarditis, and therefore a lesion of acute rheumatism ; or it is a manifestation of pyæmia.

Pathology.—Endocarditis, or indeed visceral pericarditis, is rarely limited to these membranes. The inflammatory process extends to the subjacent muscular tissue of the heart in a more or less degree. In cases of mitral endocarditis, inflammation from the valve may also involve the musculo-fibrous ring ; and if it occur before puberty, it usually leads to permanent stenosis.

The muscular fibres of the heart, whether the primary inflammation extend from the external or from the internal surface, have the same appearances as are presented in

inflammation of other muscles. The vessels are hyperæmic ; there is migration of blood corpuscles ; the cellular elements of the connective tissue and of the sarcolemma proliferate ; and the muscular striæ become blurred or obliterated. In some instances the inflammatory condition extends to the whole organ, or it may be limited to one ventricle, usually the left, or to isolated patches in different parts of the cardiac walls. The effect of the inflammation is to render the muscular tissue brittle and friable, or to transform it into a fibrous condition, either of which favours the subsequent onset of aneurysmal dilatation. Occasionally the inflammatory process goes on to the formation of abscesses, which would be small and multiple in pyæmic myocarditis, or single and of varying capacity in those forms which are secondary to rheumatic affection.

Treatment.—No special treatment is necessary or advisable. If the disease be a manifestation of acute rheumatism or pyæmia, the treatment will in reality be that which is described under those diseases. Digitalis and iron may be required, should the heart show signs of dilatation or failure of action.

HYPERTROPHY

Definition.—Enlargement of the heart, due to increase in its muscular and other histological elements.

Causation.—Any condition either within, or external to, the heart which gives rise to obstruction of the circulation will cause cardiac hypertrophy. To overcome this obstruction the heart is stimulated to excessive exertion, and more nutritive material is demanded by it. The condition is in reality on all fours with the healthy hypertrophy of the skeletal muscles seen in athletes. Therefore, although an abnormal condition in the heart, it is usually a compensatory one.

The chief cause is chronic valvular disease, hence it affects the left side most frequently. The following table gives a fairly complete list of causes.

(a) Left side

1. Valvular diseases
2. Pericardial adhesion
3. Chronic increase of arterial tension, *e.g.* Bright's disease, uræmia
4. Disturbed innervation, as in palpitation, exophthalmic goitre, abuse of tea, coffee, tobacco
5. Occupation: smiths, hammermen, athletes
6. Dilatation

(b) Right side

1. Valvular disease of the left side
2. Valvular disease of the right side (usually congenital)
3. Chronic diseases of the lungs, as bronchitis, asthma
4. Degeneration of heart walls
5. Dilatation

Pathology.—Three distinct varieties are described: (1) *Simple*, in which the walls are merely thickened; (2) *Eccentric*, in which the walls are not only thickened, but the cavities enlarged; (3) *Concentric*, in which the walls are thickened at the expense of the cavities.

Microscopically there is seen to be a definite increase in the number of muscular fibres. The walls and papillary muscles enlarge together; but in the right ventricle the muscular trabeculæ may be especially increased in bulk. The heart usually loses its cone shape, and becomes broader than long when both sides are equally affected. But when the left ventricle alone is hypertrophied, the heart becomes elongated and pointed; and, on the other hand, in excessive hypertrophy of the right ventricle, such as occurs in mitral stenosis, the apex may be formed entirely by the right ventricle.

When hypertrophy is limited to one cavity, it is a ventricle which is affected rather than an auricle; but obviously this rule will have certain exceptions, according to the various causes of the hypertrophy.

Dilatation is in most instances associated with hypertrophy.

Symptoms vary considerably, as the hypertrophy affects the left or the right heart. When the *left side* is involved, the patient complains of palpitation, precordial discomfort, but not much pain; also headache, flushings, noises in the ears, and flashes of light. The vessels become affected with sclerotic changes due to chronic arteritis, as a result of the irritation caused by the hypertrophy. Consequently the arteries are

hard and rigid; the cardiac area is bulged; the apex-beat is outside, and depressed below, the nipple; there is a slow, heaving impulse felt over a comparatively small area; dulness is increased, especially to the left; and on auscultation the first sound is long and muffled, the second loud, clear, and often reduplicated. But the heart sounds may be obscured or rendered less audible by an overlapping, emphysematous lung; or they may be altered or replaced by murmurs at the diseased valves which have originated the hypertrophy.

When the *right side* is affected, there are often no symptoms if only a requisite compensation is maintained; but when it is in excess there is cough, with distress on exertion, due to sclerosis of the pulmonary capillaries; the sternum is bulged; there is epigastric pulsation; the cardiac dulness is increased transversely to the right; and the second pulmonic sound is accentuated and reduplicated. Right-sided hypertrophy is nearly always attended by considerable tricuspid overflow.

Diagnosis.—FROM EXCITED ACTION.—This condition is temporary only, and due to some obvious cause. The action of the heart is short and hurried, and does not convey to the hand the prolonged, heaving throb of hypertrophy. The pre-cordial dulness, also, is not increased.

IN PERICARDIAL EFFUSION the percussion note is dull, resistant, and ‘tubby,’ and the apex-beat is tilted, or even lost.

IN RETRACTED LUNG the cardiac dulness is increased, but the apex-beat is normal in position and force.

Prognosis.—Hypertrophy is a compensatory condition, and, as a rule, is beneficial. So long as valvular disease exists, diminution of hypertrophy cannot be expected, nor is it desirable. The arrest of hypertrophy will depend on failure of nutrition in the heart and the general tissues of the body. Such failure may be hastened by emotion, undue exertion, and the like, and is succeeded by dilatation.

Treatment.—Apart from treatment of the valve lesion, and the cause thereof, the treatment of hypertrophy calls for nothing special. It is obvious that we must lessen arterial tension, and diminish as much as possible the tendency to cerebral congestions and possible hæmorrhages. Therefore

the bowels should be kept freely open ; alcohol is injurious, and excitement of all forms should be avoided. We may apply sinapisms, or even occasional blisters, to the nape of the neck to relieve the tension of the cerebral vessels. Digitalis, of course, is contra-indicated, except when dilatation accompanies hypertrophy. Aconite and mercury are the most useful drugs to reduce the force and tension of the pulse. Aconite should be given cautiously and in small doses (tinct. $\text{m}\nu$.).

DILATATION

Definition.—An enlargement of the capacity of one or more of the various chambers of the heart.

Causation.—As a primary lesion it is generally due to myocarditis, or to fatty and other degenerative changes in the heart muscle. In these cases the lesion may be partial, or local only, favouring the formation of aneurysmal bulgings of the cardiac walls. As a secondary change it is most frequently due to valvular disease, especially aortic regurgitation ; but it may also be caused by long-continued and excessive arterial tension, by palpitation and other excited action, or by chronic diseases of the lungs. It will thus be seen that any condition by which the blood pressure within the heart is increased tends first to the thickening and then thinning of the walls, and, consequently, to dilatation of the cavities.

The condition may also result from pericardial adhesions, or be a part of the general muscular exhaustion supervening after fevers, syphilis, &c. In all cases where it is a secondary affection it is associated with, or preceded by, hypertrophy. There is this difference, however : hypertrophy is an effort at repair, whilst dilatation is a retrograde manifestation—an overstrain or stretching.

Pathology.—It may be, indeed generally is, associated with hypertrophy of the heart walls (active dilatation), and then it constitutes merely a form of ‘eccentric’ hypertrophy accompanied by an excess of dilatation. It may also occur with thinning of the walls (passive dilatation). In any case the heart changes its shape and becomes globular ; its muscular

tissue is of a rusty tint, flabby, easily torn, and partly fatty. The walls become thinner, so as to measure only about a third of their usual dimensions. Both ventricles are usually involved, but the right may be most attenuated. The septum is generally least affected, apparently from the support it receives from both ventricles. The various orifices are dilated, and their valves become incompetent, not so much from disease of the curtains themselves as from the widening of the orifices, so that the doors do not fit the doorways. The muscoli papillares, too, by becoming stretched, assist in creating the valvular insufficiency.

Symptoms in a great measure resemble those of hypertrophy. The transverse cardiac dulness is increased ; but in cases where the cause of the dilatation is limited to the arterial side of the heart the increased cardiac dulness is entirely left-sided, the heart then being felt flapping against the chest wall in the axillary region. In right-sided dilatation the heart forms a rounded, shepherd's-pouch-shaped dulness, accompanied by pulsation in the epigastrium. The impulse is feeble and also diffused—that is to say, it is felt over a large area ; the sounds are short and irritable, the second often being decidedly tympanitic. Eventually there supervene dropsy, palpitation, dyspnœa, sleeplessness, nightmare, precordial pains, and other signs of chronic valvular incompetence with cardiac failure.

Diagnosis.—FROM HYPERTROPHY.—In this condition the systolic impulse is increased, and the extension of cardiac dulness is downwards and outwards.

FROM PERICARDIAL EFFUSION.—Here we should expect a history of acute rheumatism or some other specific disease. The area of cardiac dulness also is more extensive and pronounced, and it is more resistant to percussion, as in pleural effusion. We should also expect to hear, over some part of the heart at least, a pericardial rub.

Prognosis.—Generally unfavourable ; but much will depend on the age of the patient and the duration and cause of the dilatation.

Treatment.—Endeavour to improve the tone of the heart muscle and to promote hypertrophy. The diet should be nutri-

tious and digestible, and consist mainly of animal food ; the meals should be small, if frequent. The patient must abstain from severe exercise and avoid mental excitement. Still, gentle exercise on the flat, and fresh air may be ordered. Give iron or arsenic or other tonics with digitalis.¹ For cardialgia we may apply belladonna plaisters, or give small doses of morphia. Embarrassment of the right side of the heart will be relieved by bleeding. Apply six or more leeches over the right hypochondrium, as the liver also shares in the venous engorgement.

DEGENERATIONS OF THE MUSCULAR WALLS

FIBROID DEGENERATION may affect all the various cavities of the heart, but is more common in the ventricles. It may be caused by chronic pericarditis and any of the specific fevers. It is, however, more frequently a sequel of chronic Bright's disease, or a tertiary manifestation of syphilis.

The disease is usually limited to certain patches in the ventricular or auricular walls ; but in severe cases it may be more general. The patches are white or greyish in colour, and under the microscope show an excess of fibroid tissue at the expense of the normal muscular fibres. Occasionally, if the disease is extensive, it causes local thinning of the heart walls, and thus leads to aneurysmal dilatations.

GRANULAR or PIGMENTARY DEGENERATION is a disease the nature of which is not yet completely understood. The ventricle walls are streaked with brown, granular bodies, hence the colour of the muscle is a dull mahogany tint resembling somewhat the colour of a dog's muscles. The exact causation is not known. It occurs in people who have died of some exhausting disease, as cancer ; and we have also seen it in patients who have died of pernicious anæmia. The strength of the ventricle walls is considerably enfeebled.

FATTY DEGENERATION.—In this condition a degenerative change occurs by which the muscular fibres are destroyed

¹ R. Tinct : Ferri Perchlor : ℥x. ; Tinct : Digitalis ℥x. ; Spt : Chloroformi ℥xv. ; Aquam ad ʒj. Misc.

and replaced by fat. It must be distinguished from the fatty heart, in which there is merely a superabundance of fat on the surface, and also between the muscular fibres of the heart.

Causation.—*Age and Sex.*—It is most frequently met with in males after the fortieth year. *Debilitating and Wasting Diseases*, such as phthisis, cancer, syphilis, alcoholism, gout. *Poisons*, such as phosphorus and copper. *Structural Diseases of the Heart*, notably adherent pericardium, and obstruction of the coronary arteries, whether by clot or by calcareous changes.

Pathology.—The heart walls on section are pale or yellow coloured, and of flabby, greasy consistency. The fibres are brittle, they lose their striæ, become granular, and finally their sarcolemma sheath is filled with oil granules; the fibres thus retain their bulk and do not shrivel. The coronary arteries may be unaffected, or are atheromatous or calcified. The degenerative changes may be general, affecting the whole organ, or local only, as seen in localised patches of old pericarditis.

In fatty infiltration, on the other hand, fat accumulates in great excess between the muscular bundles, and is therefore not necessarily a condition of decay, but merely an excess of fat; and it is not till the advanced stages that any actual destruction of muscular tissue takes place, though the effects of the infiltration produce results similar to those which attend degeneration. Death may occur from syncope, or from rupture of the ventricle wall.

Symptoms.—The onset is gradual. The patient gets pale and sallow; his breathing is irregular and sighing, and occasionally assumes a 'Cheyne-Stokes' character; dyspnoea occurs on the slightest exertion, and also, at times, in paroxysms without exertion. The pulse is feeble and intermittent, and is especially irregular whilst standing or after any muscular effort. Anginoid attacks are not infrequent. In addition, certain cerebral symptoms due to anæmia of the brain centres are observed, such as giddiness, syncope, loss of memory, an irritable temper or lowness of spirits, and occasionally epileptiform attacks. Confirmatory changes are found in the eye (arcus senilis) and blood-vessels. One notable symptom in all

cases of cardiac failure from degeneration is a 'grasping' sensation at the heart, which supervenes when the patient is dropping off to sleep.

On auscultation the heart's sounds are feeble and muffled, the first sound being especially weak and shortened, and by comparison the second sound appears accentuated. The pause is abnormally prolonged; the impulse is also weak and diffused; there is scarcely any movement over the precordial region; the cardiac dulness is not increased until the organ is dilated.

Diagnosis.—It may be confused with simple dilatation, in which the dulness is increased from the first, and which improves under the influence of digitalis and other cardiac stimulants.

Treatment.—Avoid everything which tends to unduly tax the heart. Nevertheless, gentle exercise on the flat, fresh air, and healthy occupation are beneficial. Tonics, such as iron, strychnia, and quinine, are generally indicated. Digitalis may be required in advanced cases when the heart's action becomes irregular or intermittent, or shows other signs of failure.

ANEURYSM OF THE HEART

This condition may occur at any period of life from puberty to old age. Males suffer more frequently than females. It is secondary to some degenerative change by which the heart's walls are weakened, as a whole, or locally only, at various spots. Usually the apex of the left ventricle is the seat of the dilatation. The aneurysm may vary in size from an acorn to that of a small orange. It may communicate freely with the ventricular cavity by a wide mouth or only by a circumscribed and narrow, sharp-edged orifice. The sac is usually lined with endocardium, in which calcareous salts may be deposited, and it is more or less filled with a laminated or an irregular clot. As it increases in size, its walls naturally become thinner, and eventually rupture under the influence of some strain or excitement. Occasionally pleuro-pericardial adhesions delay this fatal event.

Symptoms are those which attend chronic endocarditis

and valvular disease. The aneurysm rarely attains such a size as to give rise to abnormal percussion signs, and, as a rule, the condition is only found on dissection after the patient has died from sudden syncope.

Treatment.—Whenever this condition is suspected the treatment is practically the same as in aortic aneurysm.

NEW GROWTHS AND PARASITES

Tubercle may exist in the heart either in the scattered miliary form, or as yellow, caseating masses. The pericardial surface is their most usual site ; but they may be found diffused between the muscular fibres and causing surrounding inflammation. Tubercle is never primarily deposited in the heart, but is always associated with previous deposits in the lungs or elsewhere.

Malignant New Growths.—Primary malignant growth of the heart is practically unknown. Cancer and sarcoma may secondarily involve the heart and its membranes, causing typical local nodular or flattened growths of various sizes. We know of no symptom by which their presence could be diagnosed. Their existence could only be surmised in cases in which the heart is altered in action, or displaced, or shows other signs of embarrassment in those cases in which the presence of new growth had been detected in the mediastina or elsewhere. Sarcomatous growths have a tendency to invade the heart by passing along the arterial and venous channels.

Syphilis may be manifested as gummatous tumours in the heart walls or on its surface, or as local fibrous scars and thickenings in the muscular tissue. If the growth be gummatous, it is liable to undergo the same softening and other retrograde processes which attend gumma elsewhere.

Parasites.—**HYDATID CYST** may be developed intramurally, or on the surface, beneath the pericardium. Its presence would not necessarily give rise to symptoms, nor to physical signs, until it reached such dimensions as to impede the action of the heart, or until it suppurated, or until it ruptured into the pericardial cavity.

Treatment.—Obviously no rule can be laid down for treatment in the above rare conditions beyond that which is required to alleviate the general cardiac symptoms and to mitigate or arrest the various general diseases of which the cardiac conditions are only a part.

MALFORMATIONS

It is evident when one considers the development of the heart (see p. 392) that malformations may occur in connection with the two large vessels issuing from the base, and also in connection with the auricles and with the ventricles. These malformations may result either from arrest of growth, and therefore of capacity, of these tubes and cavities, or from incomplete separation of one cavity from another.

Pulmonary Artery, and Aorta.—As regards these vessels, one or other may be constricted or actually impervious ; or they may be transposed, the aorta coming off from the right ventricle, and the pulmonary artery from the left ; or, again, the septal division between the two may be incomplete, so that there is a communication, more or less free, between the arterial and the venous blood-currents. The ductus arteriosus may also remain patent ; but here the condition is not so dangerous unless the aorta or the pulmonary artery be constricted.

The **Auricles** may retain their early fetal communication with each other, owing to incomplete development of the septum ; and seeing that the filling-up of the foramen ovale is the last completed developmental process in the auricles, and takes place after birth, we may have all degrees of imperfection from the one in which there is no, or only a rudimentary, septum, to that in which a slight valve-like slit of communication remains at the edge of the foramen ovale. Indeed, this latter condition is frequently found post mortem in patients who have died from other ailments in adult life.

The **Ventricles** in a similar way may communicate with each other, owing to incompleteness of the septum. The aperture may be large or small, according to the degree of arrested development. Usually, however, it is represented by a button-hole orifice at the upper end of the septum,

between the anterior and the right posterior aortic cusp or 'undefended space,' this part being the last to be completed. This condition is often secondary to obstruction in the pulmonary artery during foetal life, and hence the blood pressure in the right ventricle perpetuates the communication between the two ventricles.

Symptoms.—The symptoms of cardiac malformations are mainly those of dyspnoea, more or less urgent, according to the degree of abnormality, accompanied by general cyanosis, especially of the fingers, toes, nose, lips, and ears, together with clubbed finger ends, general thickening of the integument, and eventually dropsy.

The auscultatory signs are various. There are usually one or more well-defined murmurs; but their characters and intensities are subject to great differences. In some they may be booming, in others musical, or even whistling or rattling. No exact diagnosis can be made as to the character, extent, or even locality of the malformation, and any opinion on these points can only be conjectural. Exception, perhaps, may be made in cases of communication between the aorta and pulmonary artery, in which booming murmurs may be heard to attend both systole and diastole, with no interval between, the tone of the two murmurs being only slightly different.

Prognosis.—Always unfavourable. The child usually dies within a few weeks or months of its birth. Occasionally a case may survive to puberty.

Treatment.—Nothing can be done beyond keeping the patient warm, and guarding against any exertion or excitement.

EXOPHTHALMIC GOÏTRE (GRAVES'S DISEASE; BASEDOW'S DISEASE)

Definition.—A disease characterised by rapid action of the heart, enlargement of the thyroid gland, and prominence or protrusion of the eyeballs.

Causation.—*Age and Sex.*—It occurs for the most part in young adult women, and often appears in some way to be associated with uterine disorders. *Neuroses.*—It may also.

supervene in those who are overworked or who have had much mental distress and worry.

The direct cause is not yet established ; but it would seem from many of its symptoms to be due to stimulation of the cervical sympathetic, which causes vascular engorgement of the thyroid, and palpitation.

Pathology.—Certain changes, mostly in the capillaries of the cervical sympathetic ganglia, have been described. Hale-White also has found hæmorrhages in the medulla. The fat and the vascularity of the orbits and eyeballs are increased.

Symptoms.—There are three cardinal symptoms, viz. : goitre or enlargement of the thyroid body, proptosis or protrusion of the eyeballs, and palpitation. There is in many instances no definite order of onset of these symptoms. Usually palpitation is the first symptom which is complained of, and it is the most constant of the three. The pulse-rate is 100 a minute, or faster. The heart sounds are accentuated and sharp, and the cardiac area of dulness is increased.

Probably at the same time, or a little later, the thyroid body is noticed as increased in size. It is soft in consistency, and may be symmetrically enlarged, or one lobe only is hypertrophied. Auscultation over the gland detects vascular murmurs, either single, attending systole, or double, and varying in intensity and character. Sooner or later exophthalmos is observed. This may consist of only a slight protrusion of the eyeball, with some widening of the palpebral fissure, due to spasm of the muscular fibres of the upper lid, or the extrusion may be so marked as to expose the insertions of the recti muscles. The upper lids are often retracted so much as to show a clear ring of sclerotic round the cornea (Dalrymple's sign) ; they do not follow the eyeballs in their downward movements (von Graefe's sign) ; and there is incomplete and diminished frequency of involuntary blinking (Stellwag's sign). Notwithstanding the exposure there is no inflammation of the conjunctivæ, as a rule ; but the patient may become myopic. The ophthalmoscope reveals pulsations in the retinal vessels. In addition, the sufferer has other nervous symptoms. She has pigmentary patches on the skin ; she is languid or, at

times, irritable or depressed, and suffers from insomnia. She is also troubled with dyspepsia, constipation, or irregularity of the bowels, and frequently has leucorrhœa. Subsequently hypertrophy and dilatation of the heart ensue, consequent on the excessive palpitation. It should be noted that all or many of the above symptoms are liable to exacerbations and remissions.

Prognosis.—The disease is not immediately dangerous to life. Some patients survive a good number of years. Some, again, recover, or at least have been considerably relieved under appropriate treatment.

Treatment is palliative only. Tonics have chiefly been relied on, such as strychnia, quinine, and iron. Digitalis is useful in controlling the violent action of the heart. A long-continued course of belladonna has been advocated, and appears to give benefit in some instances. Give bromides for sleeplessness. The diet should be spare, but nutritious; the meals being carefully prepared and never excessive. Tea, coffee, and all other things which are liable to increase the heart's action must be avoided.

ANGINA PECTORIS (BREAST PANG)

Definition.—A disease characterised by pain in the chest, more especially in the heart region, occurring in paroxysms, and attended by pallor, cold sweats, and fear of impending death.

Causation.—(a) **PREDISPOSING.**—*Age.*—Almost invariably after middle age, especially about fifty or upwards. *Sex.*—Men suffer much more frequently than women. *Heart Excitement*, whether it be due to sudden or prolonged exertion, or to mental emotion. *Errors of Diet*, such as large meals, indigestible food, or an excess of saccharine foods. It occurs consequently in the glycosuria of ingestion (Ord).

(b) **EXCITING.**—Most cases can be included in one or other of three distinct groups, viz.: *Diseases of the Cardio-vascular System.*—It may attend any form of valvular disease, but especially aortic regurgitation. It may occur in disease of ventricle walls, such as fatty degeneration, and is then usually brought on by some exertion: the cramp or 'stitch'

of swimmers and athletes is an example. It is apt to supervene in disease of the coronary arteries, such as thrombosis and calcareous changes. It may also be caused by disease of the aorta, such as aortitis and calcareous degeneration ; by disease of the systemic arteries, especially excessive tension in the arterioles of the voluntary muscles. *Nerve Disorders*.—It is supposed to be directly due to some functional disorder of the vagus, or of cardiac plexuses, or to some other more obvious neurosis. It is, therefore, in some of these cases at least, akin to tic-douloureux, which is attended by spasm of muscle ; in others it would appear to be a manifestation of asthma or of epilepsy. *Reflex*.—It may be a reflex disorder consequent on dyspepsia, constipation, and disease of abdominal organs. But in all such cases there is a prior existing weakness of the heart.

Pathology.—Not yet thoroughly understood. It appears most probable that it is really a spasm of heart muscle, and therefore a 'heart cramp,' the spasm affecting the whole organ or a few groups of muscular fibres ; in other words a condition comparable to muscular cramp which occasionally seizes athletes during a prolonged struggle. Against this view it is stated that on post-mortem examination the heart muscle is always found to be flabby. But the spasm may be localised to part of the ventricle wall. In cramp of a voluntary muscle the spasm by no means always affects the whole of the failing muscle ; one portion only may be involved, and the pain is quite sufficient to bring the sufferer to the ground.

Another view is that the spasm is due to disease and narrowing of the coronary arteries, and consequent anæmia of the heart.

In most cases there is some organic lesion of valve or wall or of aorta.

Symptoms.—The onset is sudden, generally after some cardiac excitement, such as hurrying to the train, or severe mental emotion. The patient is seized with agonising precordial pain of a stabbing or suffocative character, extending down the left arm, or to the spine, or to the left shoulder. He has an anxious, suffering, and distressful face ; there is a

fear of impending death ; he clutches a chair, the railings, or anything at hand for support ; the body and extremities are cold, yet bathed with clammy sweats. But the mind is clear ; there is, as a rule, no syncope, and often no disturbance in the cardiac rhythm, or force. But this is not invariably so. The pulse may be one of high tension, but is quite as often slack and unaffected ; or, again, it may be irregular and intermittent ; there is no general rule.

Death may occur during the first or during a subsequent attack ; but in favourable cases the symptoms gradually subside, having lasted from a few minutes to half an hour. The severity of the symptoms ranges from slight precordial discomfort to the most severe anguish. The attacks are usually separated by long, but gradually lessening, intervals.

Diagnosis.—(1) FROM ASTHMA.—This disease is attended by cough and not by agonising pain. Stethoscopic examination of the chest also would reveal the physical signs of asthma.

(2) FROM INTERCOSTAL NEURALGIA.—Here the pain and agony are not so intense, and there is tenderness in the course of the intercostal nerves. The absence of obvious heart disease would also guide us.

(3) FROM HYSTERIA.—This mostly affects women. There are no cardiac symptoms ; the pulse is always regular ; and the pseudo-pains are associated with noise, muscular activity, and other nervous symptoms.

Prognosis.—Unfavourable. The attacks are sooner or later fatal ; indeed, a first attack may cause death, though there are usually several. Much, however, can be done to postpone them.

Treatment.—(a) PROPHYLACTIC.—Attend to the general health ; the patient should avoid undue excitement and muscular effort ; the food should be nourishing and easily digested ; alcoholic stimulants should be taken sparingly ; the bowels should act easily and without straining ; and the patient should be kept warmly clothed, especially avoiding an east wind. We may give tonics, such as iron or arsenic, to improve digestion and the health of tissues. Iodide of potassium has been advocated by Dr. Lauder Brunton.

(b) CURATIVE.—Nitrite of amyl inhalations (nij. to v.) give almost instant relief, in a majority of cases, by relaxing spasm of the arterioles. Till the drug can be procured, apply hot flannels to the precordium and give hot diffusible stimulants (brandy, ether, &c.). Nitro-glycerine (gr. $\frac{1}{100}$) is also of great service, but less so than nitrite of amyl. If, between the attacks, the arterial tension be very pronounced, good results may ensue from free blood-letting.

PALPITATION

Definition.—An increase of the heart's action in frequency and force, causing discomfort and distress.

Causation.—The condition may arise in connection with structural diseases of the heart; but, apart from heart lesions, palpitations are chiefly reflex manifestations of disorders of other organs. Thus it is associated with *Diseases* of the stomach, liver, uterus, and is found, therefore, to accompany indigestion, over-distension of the stomach, and uterine hæmorrhages. In this latter condition palpitation is most apt to supervene on lying down. *Effects of Drugs, and Dietary.*—It occurs as a result of excessive tobacco-smoking, or a too free indulgence in tea, coffee, and alcohol. *Disordered States of the Blood,* such as in anæmia, gout, scurvy, and the specific fevers. *Neuroses.*—It is frequent in hysteria, chorea, and in any condition of abnormal mental excitement.

Pathology.—Although disturbances occur in the cardiac nerve ganglia, no structural change has been described. The heart, however, after prolonged palpitation, and as a result of it, may undergo hypertrophy and dilatation.

Symptoms.—The onset is often sudden, coming on, in some individuals, whilst at rest or during sleep, but usually after excitement or exertion. The patient experiences a sense of oppression over the precordium, accompanied by dyspnœa and fainting or giddiness, with generally a fear of impending death. The heart's action is heaving, quick, irregular, or intermittent, and the impulse is diffused. The heart sounds are always accentuated, and often attended with functional

murmurs at one or other orifice. There is a marked beating of the carotids, accompanied by a rushing or whirring sound in the ears. The pulse is jerky and irritable.

These symptoms are liable to occur in paroxysms which may last from a few minutes to some hours or even days ; and they not infrequently cease as suddenly as they begin.

Treatment should be directed to remedying the cause of the disturbance. A light diet is essential, and the meals should be scanty. All indulgences in tobacco, tea, and other narcotics should be forbidden if their use is clearly associated with palpitation.

During the attack, place the patient in a recumbent position. Ether, ammonia, valerian, and digitalis are the chief remedial agents.

Marked relief can be afforded in some cases caused by extreme distension of the stomach by gas, by the passage of a stomach-tube.

MEDIASTINAL TUMOURS

The principal tumours occurring in the mediastina may be classified as those which are in connection with, or originate from—(1) LYMPHATIC GLANDS ; (2) the CONNECTIVE TISSUE ; (3) the AORTA ; and (4) the THYMUS GLAND. Other organs may be the seat of new growth or of other forms of tumours, which may extend to and involve the mediastinal spaces.

The LYMPHATIC GLANDS may be infiltrated with new growth, such as sarcoma, carcinoma, tubercle, syphilis, and lymphadenoma, but mostly as a condition secondary to deposits elsewhere. Sarcomatous tumours, however, are often primary.

The CONNECTIVE TISSUE may be the seat of acute inflammation (mediastinitis), which may or may not go on to suppuration. Inflammation sometimes, though rarely, occurs primarily, but is more frequently secondary to some injury, such as a blow on the sternum, or to inflammatory extensions from one or other of the many structures—such as lymph glands—and tubes situated in this region. Cases have been recorded in which pus has ultimately formed, and, after producing severe

pain and other symptoms which simulated aneurysm, discharged itself by the bronchial tubes.

Hydatid cyst also may develop in the mediastinal connective tissue, and attain a large size before causing any physical signs or even distress.

Hæmorrhages also have been known to occur either in connection with aneurysm or with new growth, or to be due to phthisis or other disease of the lungs. On account of the extensive amount of connective tissue in this region, it is obvious that a large amount of blood may be extravasated and yet produce no symptoms beyond those of pallor and syncope.

ANEURYSM may spring from any part of the aortic arch, or from the descending thoracic aorta, or from the innominate or the left carotid and subclavian branches. The symptoms of aneurysm of the aorta are described in a separate chapter.

The THYMUS GLAND, or the remains of it, may undergo hypertrophy, malignant infiltration, or suppuration. Such conditions, however, are extremely rare.

Symptoms.—There are no, or at least very few, symptoms which are diagnostic of mediastinal tumours, except in the case of aneurysm. The principal symptoms are those of pain, and ultimately those which are due to pressure on one or more of the various and complex structures in the chest. And remembering the anatomical relation of these structures, it will be easily apparent that these pressure signs will vary considerably, both as regards intensity and gravity, according to the site of origin of the tumour and the direction in which it grows. Thus, there may occur, at one or other period of the illness, engorgement of the veins of the head and neck and arms, accompanied by œdema of these parts. These conditions, again, may be limited to the left arm and left side of the neck, should the tumour involve the left innominate vein only; or it may probably be symmetrical in those cases in which the superior cava is obstructed. Occasionally, as showing the distributed and uneven character of the pressure, small veins here and there on the surface of the thorax are distended and varicose. This condition is frequent in multiple sarcomatous growths.

Again, as evidence of pressure on the various nerves, the patient may complain of intercostal pains, or neuralgia extending down one or both arms ; there may be paralysis of the left vocal cord ; or hiccough from irritation of the phrenics. Cough with, perhaps, bronchitis, pneumonia, or pleurisy with effusion, would point to the tumour invading the respiratory tract. In another case dysphagia with rapid emaciation may occur from pressure on the œsophagus and thoracic duct. The bones of the thorax, however, are seldom eroded by new growth as they are in aneurysm.

Other signs which should guide us are, displacements of the heart, lungs, and perhaps of some of the abdominal organs which are immediately in relation with the diaphragm. The thorax itself may be distorted, not only from the mechanical pressure of the tumour itself on the parietes, but also from collapse of a lung which it may occasion.

In order to assist us in our diagnosis, we search for new growth in connection with abdominal or other organs and glands ; we inquire if there is any syphilitic history.

Other symptoms which supervene sooner or later in all mediastinal tumours are, anæmia, dyspnœa, emaciation, and possibly paraplegia. But there is no fever except in lymphadenoma, in tuberculosis, in abscess, and in the secondary inflammations caused by the tumour.

Prognosis.—As a rule, unfavourable. The disease, whatever its nature, usually advances steadily towards death. A syphilitic tumour, however, holds out good hopes for amelioration.

Treatment can only be palliative in malignant tumours. Pain must be relieved by morphia. Chloral, on the whole, appears to be the best drug to procure sleep. It is always advisable to evacuate any pleural effusions which may from time to time accumulate. In the majority of cases in which the diagnosis is obscure, a prolonged course of iodide of potassium would be given by most physicians, with a view to promote absorption in case the tumour be syphilitic

DISEASES OF THE BLOOD-VESSELS

ARTERITIS

Definition.—Inflammation of the arteries. The disease may be acute or chronic.

Causation.—The disease may be limited to the external coat (PERIARTERITIS), to the internal (ENDOARTERITIS), or it may involve all the tunics of a vessel.

PERIARTERITIS is usually a condition secondary to injury of the vessel, or to extension of inflammation to the sheath from surrounding tissues. It may also be a syphilitic lesion.

ENDOARTERITIS may be a result of pyæmia, scarlet or other specific fevers, and also of syphilis, or of tubercle. It also occurs in chronic Bright's disease and in the general degenerative changes of the system which result from chronic alcoholism, exposures, and deficiency of food.

Pathology.—The microscopic changes which occur in an inflamed vessel are much the same, no matter which tunic is involved. The artery is thickened by hyperplasia of its cellular elements, and naturally this is most extensive in periarteritis. This overgrowth may eventually cause entire destruction of the normal histological constituents, which are replaced by a fibrous sclerosis. In endoarteritis the inflammatory process forms local thickenings in smaller or larger patches, which tend to run together, and thus produce an irregularity of lumen and an unevenness of surface. It is especially met with in the arteries at the base of the brain and in the aorta, and in many instances it is an undoubted syphilitic lesion. But it is rarely or never the only manifestation

of syphilis. The subsequent results of arteritis are seen in destruction of the elasticity of the vessels. Thus, they become hard, brittle, and tortuous; their channels are narrowed or obliterated even; and their resisting power is weakened; hence local dilatations or aneurysms are apt to supervene. Thrombosis is also a condition frequently seen in arteritis, and, although often looked upon as a cause, it is probably more frequently an effect of the disease.

Symptoms.—The main symptoms are pain, tenderness, and induration in the course of the artery. According to the extent of the lesion, these signs may be local only, or they may extend for a varying distance above and below the initial site of the inflammation. The disease is attended by fever, which, again, may vary in severity according to the area and size of the vessels affected. The inflammation may run an acute course, and perhaps terminate in suppuration, causing perforation or destruction of the artery; or, being more chronic, it gives rise to thickening of the different coats of the vessel, with permanent thrombotic occlusion of its calibre, and subsequent aneurysm behind the lesion.

Treatment.—Nothing specially can be done. Absolute rest is the first essential. Medicinal treatment consists in giving such drugs as are indicated by a syphilitic or a gouty history.

DEGENERATION OF ARTERIES

Atheroma

This is the most frequent degeneration found in the arteries. It is essentially a senile change, but may nevertheless be met with in early and middle life as a result of syphilis, rheumatism, specific fevers, alcoholism, and want of food—all producing early decay.

Its principal seat is the arch of the aorta and the arteries at the base of the brain; but no vessel is exempt from the disease. When the arch of the aorta is involved, a similar lesion is not infrequently found on the mitral valve, especially on that flap which is situated between the mitral and the aortic orifices.

The first pathological change occurs in the deeper layers of the tunica intima. It consists of an inflammatory proliferation of the cells, which, gradually increasing, causes an upheaval of the endothelial layer of cells, and thus produces a waxy-looking patch on the inner surface of the artery. Then the cellular elements undergo fatty degenerative changes; the patch becomes softened and eventually breaks through to the surface, leaving an irregular, ulcerous lesion, the floor of which is formed by the muscular and the fibrous tunics. In the last stage the abraded surface is coated with a deposit of calcareous salts intermixed with fibrin deposited from the blood.

Atheromatous disease is by no means uniform; hence the artery affected may present patches of calcareous deposit in one, and the waxy-looking patches in other parts, the surface of the vessel between the lesions often being apparently healthy.

The results of atheroma are seen in rigidity of the vessel, and a weakening of its resistance to the blood pressure. Hence the artery becomes a permanently enlarged tube with an irregular, aneurysmal dilatation, as in the aorta; or an elongated, tortuous, and hardened vessel, as in the femoral, brachial, and other arteries.

The heart necessarily becomes hypertrophied in its left ventricle, and the pulse-beat is prolonged and its recoil sudden. Anasarca of the lower limbs is an occasional accompaniment.

Fatty Degeneration

This form of degeneration, apart from atheromatous disease, has been described as affecting the arteries. It is, however, extremely rare. The fatty change may affect all the coats, but more especially the inner one. It produces much the same conditions as atheroma, and is an indication of senile decay.

Lardaceous Degeneration

This occurs as a complication of tertiary syphilis, or after prolonged suppuration. All the coats of the arteries, especially the smaller ones, and of the capillaries are infiltrated with the 'lardaceous' or 'amyloid' material. As a result,

two distinctive features are observed on microscopical examination, viz. : (1) the various tunics are with difficulty distinguishable from one another, being held together, or congealed as it were, by the degenerative albuminoid matter ; and (2) the various cells and other histological elements are obliterated or replaced by the amyloid material. Hence an affected artery presents the appearance of a hollow cylinder with a single waxy, translucent, and homogeneous coat.

ANEURYSM

Definition.—A local dilatation of an artery, with or without rupture of one or more of its tunics.

Causation.—(a) **PREDISPOSING CAUSES.**—*Age.*—Most frequent between thirty and fifty. *Sex.*—A large majority of cases occurs in males. *Climate.*—It is more prevalent in hot and mountainous districts. *Occupation.*—Sailors, soldiers, police, bargemen, ironworkers, and others whose employments are conducive to injury and to heavy muscular strains. *Disease.*—Syphilis, alcoholism, gout, Bright's disease, and all other ailments which tend to impair the health of the tissues generally. *Abnormal Condition of Vessels.*—Want of elasticity and resisting power, such as supervenes as a sequel to atheroma, embolism and arteritis, are strong predisposing causes. These conditions may be brought on by rheumatism, scarlet and other specific fevers ; or they may be the result of inflammatory changes in the vessels, and weakened support of surrounding tissues, such as occur in abscess or cavity of the lungs. *Heredity.*—Different members of the same family or of collateral branches have suffered from the disease.

(b) **EXCITING CAUSE.**—Most cases seem to date from a severe muscular strain, or violence, or direct blow. Generally we may say that there are two factors in the causation of aneurysm, viz. : a weakened arterial system with great muscular development, so that feats of endurance and efforts of strength are attempted which the circulatory system cannot bear.

Pathology.—The starting-point of all aneurysms is a weak spot in the vessel. A patch of atheroma is the most usual pathological change ; but it may originate in a general endoarteritis, the exciting cause being injury or excessive muscular strain. There are different varieties of aneurysm which are described in larger text-books. Ordinarily there is an abrupt dilatation of the artery so as to form a sac, in which some or all of the coats are found. In many cases, however, the t. adventitia alone remains, with traces here and there of the t. intima. This form is known as a *true* aneurysm, and is the one most usually met with in medical practice.

The saccular variety is a unilateral dilatation, with an orifice much smaller than the sac. All the coats of the vessel may be traced in the sac, but the t. intima is flattened and attenuated and here and there entirely wanting ; the t. media is generally deficient ; whilst the t. adventitia is distended and thickened, consisting as it does of fibrous laminae with flattened connective-tissue cells interposed. Inside, the sac is lined with laminated clot, an effort at repair. Externally, the sac blends with the tissues and structures around, which become, in fact, part of the sac. Hence in advanced chronic thoracic aneurysms we may find the actual arterial envelope almost entirely absorbed, the limiting walls being formed by surrounding tissues and organs, whether they be bone, cartilage, lung, or pleura.

A cylindrical or fusiform aneurysm consists of a symmetrical dilatation of a vessel in which all its coats are retained, the two inner being flattened and thinner, whilst the t. adventitia is distended only.

THORACIC ANEURYSM

Definition.—Aneurysm occurring in the arch of, or in the descending thoracic (dorsal) portion of, the aorta.

Aneurysm of the Arch

Symptoms.—There are five signs only which are diagnostic, viz. : (i) ABNORMAL DULNESS ; (ii) PULSATION OVER THE DULNESS ; (iii) THRILL ; (iv) BULGING ; (v) DIASTOLIC SHOCK.

(i) **DULNESS** will vary in situation and extent according to origin and size of the sac. Aneurysms of the first part of the arch tend to extend upwards and to the right of the sternum ; those involving the second, or oblique portion, usually extend backwards and upwards ; while those springing from the third portion increase towards the left lung, or inwards towards the heart and vertebral column.

(ii) **EXPANSILE PULSATION**, in combination with abnormal dulness, is almost diagnostic. The systole of the heart is imparted to the aneurysm and is generally easily felt by the hand, except in deeply seated dilatations. The ear, however, aided by a rigid stethoscope, will, as Dr. Douglas Powell has stated, nearly always detect this sign. Pulsation in the sac may be imparted to the trachea or large tubes producing a puffing character of expiration, or it may cause gurgling sounds (synchronous with the heart's action) in the œsophagus during drinking.

(iii) **THRILL** is a valuable diagnostic sign ; but is not always present. It is best marked in sacciform dilatations which contain little or no clot.

(iv) **BULGE** may be absent entirely if the aneurysm does not come to the chest parietes. It may, however, give rise to a tumour, varying in size from a small egg to that of a child's head, or even larger. When it occurs in the region of the costal cartilages it is obviously more abrupt and localised than when the ribs only are involved. The size of the bulging is considerably diminished after death.

(v) **DIASTOLIC SHOCK** is one of the most reliable signs. It occurs immediately after the ventricular systole, and is caused by reflux of blood from the distended aneurysm accentuated by the elastic recoil of the vessel and surrounding structures. It may therefore be looked upon as the systole of the sac, occurring contemporarily with the diastole of the ventricle ; and it becomes weaker as the sac's walls become thinner.

Pressure Signs.—The other signs are those of pressure on surrounding structures, and are common to new growth, abscess, hydatid cyst, and all thoracic tumours, as well as aneurysm. They may be classified on an anatomical basis according to the different structures involved. Thus—

(a) BONE.—Erosion of bodies of vertebræ leaving the intervertebral cartilages intact ; the bone resists and is absorbed, whilst the elastic discs yield and are preserved. Erosion and absorption of sternum, ribs, or costal cartilages may also occur.

(b) DIGESTIVE TRACT.—Causing dysphagia, and perhaps ulceration of the œsophagus, into which the sac may rupture. Rapid emaciation occurs if the thoracic duct be involved, but this complication is rare.

(c) RESPIRATORY TRACT.—Flattening or displacement of trachea or bronchi, tracheitis with tracheal stridor and deep rhonchus ('leopard's growl'), erosion of bronchial cartilages, bronchitis with profuse expectoration (the most frequent respiratory complication), pneumonic consolidation, gangrene of lungs, and pleurisy. A harsh, clanging cough is also present at advanced stages of the disease.

(d) ARTERIAL SYSTEM.—Cardiac hypertrophy and aortic incompetence are the most common. The regurgitation is not always due to valve disease, but is often caused by the sac extending downwards to the aortic ring and so rendering the valve insufficient. Dilatation of left ventricle, dissimilarity of pulses, owing to obstruction of innominate or of left subclavian artery, are also frequent.

(e) NERVOUS SYSTEM.—Obviously the nerve pressure signs will vary according to the direction of growth of the aneurysm. Thus, a patient may complain of pains in the courses of intercostal nerves or down the inside of the arm (intercosto-humeral). There may also occur at different periods alterations in force and frequency of heart-beat (vagus), variations in character of voice from paralysis of vocal cord (left recurrent laryngeal), asymmetry of pupils (sympathetic), asthmatic dyspnoea, or hiccough (phrenic).

In reference to the pressure signs of vagus and of sympathetic, it may be pointed out that the symptoms will vary according as the pressure exerts a stimulating or a paralytic effect, and, therefore, that such signs as unequal size of pupils may be and often are misleading so far as diagnosis of the situation of aneurysm is concerned.

(f) **VENOUS SYSTEM.**—Engorgement of superficial veins of chest, arms, and head and neck. The extent of the venous obstruction and œdema will vary according as the superior cava or the innominate veins are involved. Thus, if the aneurysm springs from the first part of the arch and compresses the superior cava, the engorgement would extend to both sides of the head and neck and to both upper extremities; if it arises from the second portion, the left arm and the left side of the head and neck would most likely be œdematous; whilst aneurysm of the third portion might exist without any venous signs whatever. Ulcerative communications may occur between a trunk vein and the aneurysm sac, causing a booming or purring murmur continuous during systole and diastole.

There is no regular priority or order of pressure signs in a thoracic aneurysm, since they depend upon the site of origin and the line of extension of the sac. As a rule, aneurysm of the first part tends to extend upwards and to the right, and is attended by early aortic regurgitation, and sooner or later by venous œdema. Aneurysm of the second portion generally extends backwards and has laryngeal symptoms as the most marked feature. In the third portion the symptoms may be most obscure, but there is generally a history of pain in the mid-dorsal region and of dysphagia. A notable feature in all cases is the remission and fluctuation of symptoms. Signs of pressure on a certain structure may disappear entirely for a time, to be succeeded by pressure on a totally different organ.

Prognosis.—The disease is almost always sooner or later a fatal one. The termination of a case may result in (i) **RUPTURE** externally through the chest walls, or internally into pleura, pericardium, œsophagus, bronchial tract, or other outlet, with immediate death. Rupture into the pericardium, however, is often preceded by a warning pericarditis. Experience also shows that it is the smaller aneurysms which rupture. (ii) **CURE**, by the formation of a laminated clot filling the sac (rare). (iii) **DEATH** from exhaustion, due to pain and to pressure on nerves or other structures.

Treatment.—The chief aim is to give rest to the body, and also to the heart, so as to prevent further enlargement of the sac, to obviate its rupture, and to favour clotting. Therefore the patient should avoid exertion, excitement, and severe muscular efforts. The diet should be spare, unstimulating, and free from alcohol if possible. Spirits especially appear to produce an irritating effect. By these means we hope (i) to reduce the number of the heart-beats *per diem*; (ii) to diminish the blood volume passing through the sac; (iii) to reduce the force of blood pressure and current in the sac, and so favour gradual blood-clotting. With these points in view Tufnell advocated perfect rest in bed, aided by sedatives if necessary, for six months; daily relief of the bowels by laxatives; and a restricted dietary consisting of ten ounces of solids, with eight ounces of fluid *per diem*, divided into three meals. Such treatment, however, is tolerated with difficulty only. Balfour advocates iodide of potassium (grs. v. to xx. t.d.). This may be tried along with the Tufnell treatment. The use of the drug is especially indicated in syphilitic cases. Ice-bags may be applied when the sac is pushing its way to the surface, but the weight is often intolerable. To surgery we are indebted for heroic treatment, such as puncturing the sac with insulated needles and passing an electric current through it; or thrusting iron wires or needles or horsehair into the sac with a view to favour coagulation; or ligature of one or more of the primary vessels springing from the arch. But so far as we know these procedures hasten the fatal event. Exception, perhaps, may be made in favour of ligature of vessels, which appears to have prolonged life in some few instances.¹ Wooldridge advocated the injection of fibrinogen into the sac, and his arguments were based on sound scientific experiment. Ergot of rye yielded good results in Dr. Sibson's hands. Pain may be relieved by opium, belladonna, or by bleeding.

Seeing that an estimate of the probable duration of life in cases of aortic aneurysm is necessarily so difficult, it is an open question whether in the case of a bread-winner it were not

¹ See Barwell, *International Encyclopedia of Surgery*.

better to advise the sufferer to follow a light occupation as free from excitement as may be, and at the same time to take such precautions in diet and mode of life as are most conducive to avert the unfavourable ending.¹

ABDOMINAL ANEURYSM

Definition—Aneurysm occurring in the course of the abdominal aorta or its branches. The following description applies to the abdominal aorta only.

Causation.—Nothing beyond the usual causes common to thoracic aneurysm (p. 455), except, perhaps, certain occupations, as carpenters, shoemakers, farriers, who are prone to receive injuries to the abdomen.

Signs.—The shape of the abdomen is normal, and there is no bulging unless the aneurysmal sac be very large; nor is there dulness as a rule, the sac being overlapped by resonant bowel. A tumour can usually be felt over, and in the length of, the abdominal aorta, and therefore in the epigastrie or in the upper umbilical region; it pulsates, is expansile and mobile, and, as proving its connection with aorta, it still retains these characteristics when the patient rests in the knee-elbow position.

The aneurysm is attended by a deep-seated pain, as a rule extending from loins to thighs, groins, and abdominal walls (branches of lumbar plexus). It may be further complicated by nausea, vomiting, or distress after food (pressure on stomach), by jaundice (obstruction of common bile duct), by pain after, or difficulty in, micturition (pressure on bladder), by scanty urine (pressure on ureter), by œdema of lower limbs (obstruction to inferior vena cava), or by rapid emaciation (pressure on thoracic duct). A systolic thrill is usually present, and auscultation detects a murmur synchronous with the expansion of the sac. It is necessary to remember that a murmur may be produced in health by compressing the aorta with a rigid stethoscope. When the thigh is elevated the femoral pulse often assumes the 'water-hammer' (collapsible)

¹ See *Medical Society's Transactions*, vol. xiii.

type, or presents comparative weakness as compared with the radial pulse.

Diagnosis.—(1) *Aneurysm of Cœliac Axis.*—The tumour is situated very high up, far above the level of the xiphoid cartilage. It would cause circulatory disturbances in stomach, liver and spleen, and be attended by difficulty of breathing, radiating girdle pains, and other signs of pressure on diaphragm and its crura. Its existence can rarely be diagnosed until the sac assumes large dimensions.

(2) *Aneurysm of Superior Mesenteric.*—Pulsation ceases in the sac and in the iliac vessels on compressing the aorta above the tumour. The tumour is movable before adhesions to neighbouring structures have taken place. The common iliac vessels move on pushing the sac away from the middle line. Aneurysm of the superior mesenteric is extremely rare.

(3) *Tumours of Mesentery.*—Such as malignant or benign new growth or tubercular mass. Here the pulsation is not expansile; there is usually no bruit; and the growth of the tumour is more rapid than in aneurysm.

(4) *Uterine Tumours*, including pregnancy, may simulate abdominal aneurysm. They are, however, solid; they grow from the pelvis; and their nature would be diagnosed easily by vaginal examination.

(5) *Abnormal Pulsation and Phantom Tumours* occur in hysterical women. A similar pulsation would be found in the carotids. Phantom tumours vary in size from day to day; they disappear under an anæsthetic; and they are attended by tension of the abdominal walls.

Treatment.—(1) **PALLIATIVE.**—Rest and a restricted diet should be enforced. The bowels should act daily, and without effort. (See Thoracic Aneurysm.)

(2) **CURATIVE.**—We may try the effects of pressure on the aorta above the sac, by means of a tourniquet, or a graduated weight, or by the fingers. The bowels should be freely emptied beforehand. Care should be exercised against undue pressure which might injure the gut and other structures. We may further try the effects of prolonged rest, with large doses of

iodide of potassium (gr. xv. t.d.), or combine these means with restricted diet (Tufnell). (See Thoracic Aneurysm.)

THROMBOSIS

Definition.—Coagulation of blood within the heart or vessels, during life.

Causation.—The real cause is the formation of fibrine by the union of fibrinogen and paraglobulin in the presence of the fibrine ferment, the paraglobulin and ferment being set free only by the destruction of the white corpuscles. It is, however, necessary that the blood should be in contact with dead matter, or with altered living tissues. The exciting causes may be grouped under three headings, viz. :

1. STAGNATION OF BLOOD, as in general weakness, exhaustion after fever, or in the uterine veins after parturition.
2. INJURIES TO VESSELS, such as ligation, puncture, bruising.
3. BLOOD CHANGES, as in pyæmia, septic and other fevers.

The change may occur in the ventricles or other cardiac cavities, or in an artery, capillary, or vein. A favourite site is in such parts of the circulation where the current is sluggish or forms eddies and 'backwaters,' as among the muscoli papillares at the apex of a ventricle, and the pouches behind the venous valves.

Pathology.—Four distinct varieties of thrombus are recognised.

1. RED THROMBUS, which forms whenever a portion of blood is cut off from the circulation, as in an artery immediately above a ligation. Here the whole constituents of the blood are concerned in its formation.

2. WHITE THROMBUS, formed by the adhesion mainly, if not entirely, of white corpuscles to the inside of a vessel at the site of some injury. The leucocytes adhere to the surface of the vessel which is roughened by the injury, and are increased in quantity by successive waves somewhat similar to the augmentation of a snowball. Subsequently all traces of the

corpuscles disappear, or at least only a few may be seen ; the resulting mass being a fibrinous coagulum due to the disintegration of the corpuscles and the deposit of fibrinogen from the blood plasma.

3. MIXED THROMBUS, in which a sufficiency of ferment exists to form a coagulation of the entire blood. It is therefore red with alternation of deposits of white coagulum.

4. HYALINE THROMBUS, resulting, apparently, from pressure on the coagulum, which is thus converted into a white structureless plug adhering to the vessel walls, without any definite limiting boundary.

Changes and Subsequent Results.—(a) *Stain*.—The coagulum may become tinted brown from infiltration of colouring matter washed out from the blood corpuscles. (b) It may *soften*. This change commences at the centre, which liquefies into a dirty brown juice, consisting of debris of the thrombus. This softening extends centrifugally until ultimately a cyst may form, which, bursting, would discharge its contents into the circulation. (c) It may *shrink*, and become hard from the deposit of lime salts (phlebolith). (d) It may become *organised*. The thrombus is disintegrated by round cells, probably of inflammatory origin, and at the same time it is traversed by capillaries which spring from the vasa vasorum of the containing vessel. Thus the clot is formed into a vascular body which, subsequently developing into connective tissue, contracts and permanently obliterates the lumen of the vessel.

The chief results of thrombosis may be, (1) *Embolism* from detachment of a fragment which, being carried into the circulation, is arrested by some distant vessel. (2) *Phlebitis*, which is local, but often of severe character, causing adhesion of the vessel to its surrounding tissue. (3) *Passive congestion and œdema* of parts behind the obstructed vein. This does not usually occur until the later stages. (4) *Gangrene*.—This is a somewhat rare result, and occurring in elderly subjects is usually associated with a feeble heart and diseased arteries.¹

¹ See Coats's *Manual of Pathology*.

EMBOLISM

Definition.—The obstruction of the lumen of an artery or of a capillary by some migratory foreign body.

Causation.—Any solid matter circulating in the blood, whether it be (1) a detached portion of a thrombus, or valvular vegetation; (2) a parasite; (3) a piece of new growth; or (4) a particle of cretaceous matter. Air even, although not coming within the definition of a solid, may also act in a similar mechanical way. The introduction of air may take place through the uterine sinuses after parturition, or by operations in the neck, involving the jugular or other veins. Fatty embolism also occasionally occurs after operations on adipose tissues, or subsequent on injury to bones, or as a complication of diabetes. The dissemination of pyæmia, and other septic diseases, must also be regarded as embolic.

Pathology.—The embolus derived from one or other of the above sources is found in an artery or in a capillary. The chief exception occurs in the portal vein, which is practically an artery in its method of distribution. Further, it is mostly arrested at the bifurcation of a vessel; it may have passed readily through a primary artery, but could not pass one of its divisions. It not infrequently moulds itself astride the bifurcation, or it may be divided into two portions, one for each subdivision.

Changes and Subsequent Results.—The changes which occur after embolism are not so much seen in the obstructing body itself as in the immediate vascular zone. Thus, (1) on both sides of the embolus thrombosis takes place, the clot extending usually as far forward as the nearest distal branch and as far backwards as the last proximal branch. (2) As a result of this obstruction, an effort of repair is seen by the production of increased pressure in the collateral circulation, which may be sufficient to carry on the nutrition of the part. (3) If, however, the collateral arterial pressure be insufficient, infarction occurs on the distal side of the embolus.

This infarction is necessarily wedge-shaped, owing to the pressure on the distal side of the obstructed artery being reduced to such a degree as to allow the venous blood to extend backwards into the capillary tree which is the termination of the plugged vessel, and also to permit of free access of blood from surrounding vessels.

The capillaries, thus deprived of fresh blood, lose their nutrition, and also allow free diapedesis and hæmorrhage, the surrounding parts being infiltrated, forming a *hæmorrhagic infarction*. Subsequently, sloughing results, partly from pressure of the extravasated blood, and partly from want of nutrition.

Occasionally, as in the spleen, necrosis occurs at once, before collateral engorgement has had time to take place. The infarction then is pale, it still retains its wedge shape, and eventually contracts and cicatrises (*white infarction*).

Examples of restitution of circulation are seen in obstruction to arteries of the limbs and muscles, in which collateral anastomoses are free, and the results not generally serious.

In 'end' arteries, however, in which anastomoses are scanty, or even absent, there is no restitution of the circulation, and the results are correspondingly serious. 'End' arteries are seen in the brain, spleen, kidney, pulmonary and portal veins, retinal artery, &c. (See Coats, *op. cit.*)

RAYNAUD'S DISEASE

Definition.—A local asphyxia and symmetrical gangrene of the extremities.

Causation.—*Age.*—It is most common in children or in early adult life, between eighteen and thirty. *Sex.*—An excessive preponderance of cases are females. *Season.*—The disease prevails in cold, damp weather, and is comparatively rare in summer. *Constitutional Defects.*—The majority of cases are either feeble and ill-nourished children, or adults with defective circulatory systems, whether it be enfeebled ventricular action, or some fault or weakness in the blood-vessels themselves.

Pathology.—The sites of the chief pathological changes are, the tissues on the terminal phalanges, or on the nose, external ears, and other exposed parts which are furthest from the circulatory centre. The actual pathology is still unsettled. By some the disease is regarded as (1) a peripheral neuritis. But against this must be urged the fact that all gangrenes are attended by inflammation of the nerve-endings in common with the other tissues of the part; further, if the primary lesion be neuritis we should expect it to be chronic and persistent in its duration, regularly painful, and eventually causing some trophic lesion other than local death. (2) Some again regard the disease as a local arteritis. If this be so, it would be very unusual for such a lesion to disappear and be apparently cured in one or two days. (3) Others look on the changes as being due to spasm of the arterioles. This view commends itself to us. It would appear that there occur, in the course of the disease, local storms which have centres of greatest intensity over the fingers, toes, ears, and nose, producing damage which may be repaired, the tissues withstanding even the renewal of outbreaks on the morrow or at subsequent dates; on the other hand, the damage caused by the outbreak may be so great as to amount to gangrene and loss of parts. This view is confirmed when we note that the gangrene spares, or at least comparatively rarely attacks, the thumbs and great toes, which are extremely well supplied vascularly, and also the outer sides of the little toes and inner sides of the little fingers, all of which have a separate and undivided digital artery.

Symptoms.—The chief symptoms consist of a symmetrical gangrene of the toes or fingers.

It must not be understood that in every case the conditions run on to gangrene. They frequently, and in the early stages always, stop short of local death; indeed the stages may be variously described as local syncope, followed by local asphyxia, and subsequently by gangrene. The other chief characteristics of the disease are its symmetry and its paroxysmal recurrences.

The disease is frequently ushered in with yawning or

vomiting; the extremities tend to become cold or blue; hæmoglobinuria of an intermittent type is of common occurrence. Then the fingers or toes, the ears or nose, become dead and cold, this condition lasting from a few minutes to some hours. Subsequently to the anæsthesia the fingers are livid and cyanosed; and ultimately, as stated above, gangrene may occur.

But the anæsthesia and the cyanosis are not necessarily permanent; they are usually fugitive in character—here to-day and gone to-morrow. A finger which to all appearances was about to lose its terminal bone, nail, and skin, may on the next visit show little or no trace of the threatened local death; the disease did not go beyond temporary asphyxia in this case. Nor is the discoloration permanent: that is to say, the circulation is only stagnant; there is neither ecchymosis nor diapedesis of blood-cells to cause permanent darkening of the skin. The pain, however, whilst there is discoloration, is usually exquisitely acute.

The gangrene is not extensive; it is, as a rule, limited to the skin and tissues in relation with the ungual phalanges, or to the cartilages of ears and nose and skin over them. Occasionally, however, it does extend to the deeper structures, or it may travel backwards so as to cause the destruction of a hand, foot, nose, or ear.

The parts which are affected are, as stated, the toes, fingers, ears, and nose; but other localities may, though rarely, be affected. It is this partiality for certain exposed situations which makes us pronounce for the vascular theory of the disease. Besides, its symmetrical tendency is remarkable. If one toe is affected, not only is its opposite fellow involved, but often to the very same degree; a similar remark applies to the fingers and to the ears. The little fingers and toes, the thumbs and great toes, often escape, for anatomical reasons probably.

Occasionally during the attacks there is effusion into one or more of the larger joints, suggesting the association of the disease with rheumatism.

Should the local asphyxia go on to gangrene, it is generally

completed in about ten days. On the other hand, if the disease stops short of gangrene recovery is usually ushered in by a sort of crisis which is attended by the passing of much urine containing oxalates in large quantities.

Treatment.—Almost entirely expectant. Relieve the intense pain by opiates ; Nature must do the rest. The application of electricity to the affected parts has been advocated, and results so far seem to have been fairly good. Immerse the limb in a basin of salt and water ; place one pole of a constant current battery on the upper part of the limb and the other pole in the basin, thus converting the salt water into an electrode. Use as many cells as can be tolerated, and 'make and break' contact so as to get frequent contractions of the limb.

Shampooing and massage have also been advocated. Nitrite of amyl also has been prescribed with a view to relax the spasms of the arterioles.

If gangrene actually occur, and is superficial only, the expectant treatment is best. The parts should be left alone, except that they should be covered with cotton-wool and shielded from the air. If the gangrene extends deeper, amputation of digits, or even of limb, may be necessary.

[See Raynaud's 'Thesis,' with Dr. Barlow's appendix, New Sydenham Society, 1888 ; also the 'Lancet,' vol. i. 1887.]

PHLEBITIS

Definition.—Inflammation of the veins.

Causation.—Usually a secondary affection. (1) *Gout*.—It occurs in gouty subjects, and is then associated with either gouty deposit in one or more joints or other manifestations of gouty diathesis. (2) *Rheumatism* may similarly act as a predisposing cause. The phlebitis is then often symmetrical, and shows a tendency to metastasis. (3) *Pyæmia*, from embolic infection. (4) It occasionally supervenes as a complication after *Parturition*. (5) *Inflammatory Extensions* from neighbouring structures, such as occurs in abscesses, new growths

and the like. (6) It may be directly caused by punctures or other *Injuries* of a vein.

Pathology.—In nearly every instance thrombosis or a periphlebitis is a precedent condition. The inner coats of the vein become thickened and roughened by the inflammatory process, which thence extends to the outer coats and the surrounding sheath and connective tissue. As a result of the inflammation a clot forms, which increases longitudinally and extends backwards to the last pair of valves or to the last collateral branch. It also usually extends forwards to the next tributary, so that the conditions and subsequent changes are the same as those described in Thrombosis.

Symptoms.—Inflammation of a superficial vein is revealed by the presence of a red, hard, knotted and cord-like swelling in the course of a vessel, attended by more or less febrile disturbances according to the extent of the lesion and the size of the vein affected. Other symptoms which supervene are, œdema of the limb or of the parts below the obstruction, and possibly the formation of abscess immediately around the inflamed vessel. Suppuration would be revealed by local fluctuation and redness, with an intermittent temperature. Subsequently the clot may shrink and, adhering to the vessel, become organised; or it may break down, and allow of detached particles becoming liberated and causing embolic obstruction in the pulmonary artery or some of its branches.

Treatment.—Rest is essential. Firm and equal pressure should also be applied over the vein by means of a flannel or an elastic bandage. All liniments and rubbings should of course be forbidden, for fear of dislodging the clot. Carbonate of ammonia may be given in large doses, with a view to retard coagulation.¹ When this has once formed, iodide of potassium may promote absorption; but iron tonics are probably more useful. Any gouty or rheumatic condition may require the special treatment of those diseases.

¹ R. Ammon: Carb: gr. x.; Tinct: Lavand: Co: ℥xv.; Aquam ad ʒj. Misce; fiat mist. effervesceus cum Acid: Citrici gr. x.; Quiniæ Sulph: gr. j.; Aquam ad ʒj.

DISEASES OF THE BLOOD

PURPURA

Definition.—A primary disease of the blood-vessels or of the blood, characterised by subcutaneous and other hæmorrhages.

Causation.—(1) *Condition of Life.*—It is nearly always associated with want of exercise, or poverty and unwholesome dwellings, such as damp, underground rooms, but it is not necessarily due to deficient quality of food. (2) *Some Constitutional Defect*, such as inherited syphilis, scrofulous diathesis, &c. (3) *Neuroses.*—Fright, anxiety, mental depression.

Pathology.—The blood is thin and poor in quality, the fibrin being diminished, and the tendency to coagulation consequently diminished. Hæmorrhages similar to those on the surface may be found in the internal organs.

Symptoms.—The disease has generally some premonitory symptoms which are often vague and indefinite. They mostly consist of lassitude and loss of appetite. Then the characteristic signs of subcutaneous hæmorrhages appear. These assume various forms and degrees of severity. They may consist of minute petechial spots, which appear in successive crops, each passing through the different stages of discoloration common to bruises. Any part of the body may be subject to the hæmorrhages, but they are most frequent in the subcutaneous tissues of the legs and feet. The hæmorrhages may also be deeper seated, and show themselves as large blotches of discoloration which gradually come to the surface as dusky red patches, tender to the touch and raised above the level of

the skin. They are not infrequently caused directly by pressure or injury. Similar extravasations of both varieties may be observed under the mucous membranes, conjunctivæ, and other vascular tissues ; or there may be actual breach of surface and escape of blood from the respiratory, digestive, or genito-urinary tracts. The disease is liable to remissions and exacerbations. Death may occur, after repeated attacks, from apoplexy or from syncope as the direct result of the hæmorrhages.

Varieties.—(1) *Purpura Simplex*.—The mild form, in which the hæmorrhage is limited to petechiæ, which appear in crops. (2) *P. Hæmorrhagica*, in which hæmorrhages occur in large amounts subcutaneously, or in the submucous tissues, or into cavities and organs. It is often rapidly fatal from exhaustion. Other varieties are mainly based on differences of degree and locality of hæmorrhages, and are symptomatic only.

Diagnosis.—FROM SCURVY.—This disease is attended by swollen condition of the tongue and gums, with much fœtor. The hæmorrhages also are much more copious, and often produce large swellings or subcutaneous tumours.

Treatment.—The main indication is to improve the general health and the tone of the vascular tissues generally. A mild aperient should first be given. Diet is important. It should consist of light, nourishing, digestible food ; fruit may be freely added, such as oranges, lemons, &c. Iron and arsenic are of service as blood tonics. Ergot with opium may be prescribed if the hæmorrhage be profuse. When practicable remove the patient to the seaside or to some bracing atmosphere. Patients coming from poor, unhealthy surroundings respond to treatment so much more readily than the rich and well-to-do.

SCURVY (SCORBUTUS)

Definition.—A blood disease due to the want of fresh vegetables, and characterised by intense debility with anæmia, together with hæmorrhages from the gums and into the muscles or subcutaneous tissues of thighs, or other parts of the body.

Causation.—*Age.*—Common in old people. *Climate.*—It is more prevalent in cold, wet regions, but by no means confined to these localities ; cases have occurred in the tropics. *Occupation.*—Sailors, lumbermen (Canada), and those whose occupations entail exposure to cold and wet, with a dietary of salt- and cured meats. *Mental Depression*, as in a defeated and retreating army, a waterlogged ship, famines, &c., with their attendant discomfort and want of proper food.

The exciting cause appears to be an absence of fresh vegetables, together with a defect or excess of animal food, whether it be salt-cured or putrid.

Pathology.—The blood is pale, thin, the specific gravity diminished, but the fibrin is increased. Examination of the various organs and cavities reveals congestions, extravasations, and exudations. No organ appears to escape, hence there is a tendency to rapid decomposition. The hæmorrhages may be of different dates in the several parts.

Symptoms.—The disease is usually ushered in with premonitory pain in the back and limbs, together with great lassitude and prostration. The patient's face then becomes sallow, anæmic, and puffy ; he suffers from palpitation and dyspnoea. He also has deep-seated pains, especially in the hams, the bends of elbows, extensor surfaces of legs, the palms, and in the deep tissues of the neck, which are succeeded by local swellings due to hæmorrhages corresponding to the site of the pains. In addition there appear petechiæ or ecchymoses, mostly at the roots of hairs over the hamstring muscles, or on the extensor surfaces of legs and arms. Similar blotches of extravasation appear on the trunk, face, and other parts ; they are irregular in outline, firm or tough to the touch, and painful as in a bruise. The gums become spongy, recede from the teeth, and easily bleed ; the tongue is red, raw, and swollen ('bullock's liver') ; the breath is very foul ; and the bowels irregular, alternating between constipation and diarrhœa. The stools also are very offensive, and they may contain blood. Hæmorrhages are, in addition, liable to occur into the peritoneal, pleural, or pericardial cavities, or from the genito-urinary tract (kidney, bladder, uterus). Exhaustion

increases with the disease ; sloughings take place ; the teeth become loosened and fall out ; old cicatrices reopen, and slight abrasions of skin become unhealthy sores. Vomiting and diarrhœa may supervene ; the pulse becomes more feeble and slow ; and death occurs from exhaustion or from cerebral hæmorrhage.

Complications.—Chiefly such as are due to extravasation of blood ; hence pneumonia and apoplexy of lung, pleurisy with hæmo-thorax, cerebral effusion, coma, albuminuria, &c.

Treatment.—Cleanliness, warmth, and good food are essentials. Procure fresh vegetables as soon as possible. The best antiscorbutics are potatoes, yams, and also those vegetables which belong to the botanical order *Cruciferae*. Fruit also may be allowed (lemons, grapes), or, failing these, pickles, lime-juice.

Prevent any bruising ; treat superficial hæmorrhages on ordinary surgical principles. For hæmorrhage from the bowels give mineral acids (with opium if necessary) or tannic acid.¹ Diarrhœa may also be checked by similar treatment. The condition of tongue and gums may require astringent or deodorising washes, such as decoction of cinchona, or Condyl's fluid.

ANÆMIA

Definition.—A general term widely applied to all blood diseases in which there is deficiency in quantity (anæmia proper) or a defect in quality (spanæmia).

Causation.—Any attempt at rigid classification of the various forms of the disease is difficult. Experience shows that differentiations are not so well marked in many allied diseases, and that a given case of anæmia may resemble in many of its important features one or two other varieties ; in other words, certain distinct diseases called anæmia may overlap, not only in their symptomatology, but also in their causation. The tabulation into (a) *Primary* and (b) *Second-*

¹ R. Tinct : Opii ℥x. ; Acid : Sulph : Dil : ℥xv. ; Aquam ad ʒj. Misce.

ary, of Fagge, Osler, and others, appears on the whole best : that is to say, in some instances anæmia is an essential or *primary* disease of the blood, in others it is a symptom only, and therefore *secondary*. No hard and fast line, however, can be drawn ; and some forms of anæmia are here included as primary diseases which are not so recognised by many authorities.

The following table includes most of the causes :

(a) <i>Primary Anæmia</i>	(b) <i>Secondary Anæmia</i>
Pernicious anæmia	Leucocythæmia
Chlorosis ?	Lymphadenoma
Starvation	Excessive discharges, <i>e.g.</i> hæmorrhage, diarrhœa, leucorrhœa, polyuria, suppuration, lactation
Occupations and conditions, <i>e.g.</i> miners, clerks, convicts	Chronic diseases of stomach, spleen, kidneys, liver, heart (aortic regurgitation, pericarditis)
Senility	Specific diseases, <i>e.g.</i> rheumatism, syphilis, malarial, typhoid fever
Transition from youth to manhood	Poisons, <i>e.g.</i> mercury, lead
	Parasitic invasion, <i>e.g.</i> bilharzia hæmatobia, anchylostomum duodenale

General Symptoms.—In all cases the countenance is pallid, the mucous membranes colourless, the face and eyelids puffy, and there is a tendency to œdema of the ankles and feet. The digestive organs are out of order, and the appetite is depraved. In addition the circulatory organs are affected. The pulse is quickened ; the heart is irritable ; palpitation is frequent ; a soft, systolic, blowing murmur is heard, loudest generally over the area of the pulmonary artery, but at times diffused. A venous hum, continuous during systole and diastole, is also heard at the root of the neck, especially on the right side.

Treatment.—We must first remove all predisposing causes. If the anæmia is of malarial origin, quinine or arsenic is indicated. Mercury in small doses may be given in syphilitic cases. Some preparation of iron will be ultimately required, but the digestive organs and bowels must be in a healthy state first.

Ferr. redact. (grs. i.-v.) may be given with food ; the phosphate of iron is also of distinct advantage. The 'proto' salts, however, are generally more efficacious than the 'per' salts.

The natural chalybeate waters are usually very efficacious, and particularly those which contain manganese also.

Pernicious Anæmia (IDIOPATHIC ANÆMIA)

Definition.—A disease characterised by intense progressive pallor due to deficiency of red corpuscles, occurring mostly in adults, and steadily advancing to a fatal issue.

Causation.—The majority of cases are females. The disease is caused by pregnancy, mental shock, and other nervous disturbances ; excessive loss of blood, as after surgical operation, parturition ; intestinal parasites. It will here be seen that it is in many instances a secondary anæmia ; but cases occur in which no predisposing cause can be traced, except that there is often a previous history of chlorosis in earlier life.

Pathology.—The blood is diminished in quality and in quantity. The red corpuscles may dwindle to one-half or even one-fifth of their normal quantity. The remainder are small, ragged, and granular ; the hæmoglobin is diminished, but still relatively increased, this being due to the great destruction of cells. They therefore represent a dwindled colony, its numbers diminished, but the remnants relatively rich. The destruction of the corpuscles goes on in the liver and spleen, their pigment being deposited in the liver and other organs, and also appearing in the urine. Hæmoglobin is very readily crystallised (Copeman).

The body is not emaciated, but thickly covered with a characteristic yellow fat, the colour of which is not unlike a duckling. It is quite easily recognised when once seen. The muscles of the body are dusky red, like a dog's.

Symptoms.—Pallor which resists ordinary treatment, and which is not apparently due to any new growth, or to disease of any organ, is the principal symptom. This is followed by the usual phenomena common to any form of profound anæmia, viz. dyspnœa, epistaxis, with retinal and other hæmorrhages, gastric

disturbances, and hæmic murmurs over the heart and at the root of the neck. The temperature is noteworthy; it is irregularly intermittent, one day being pyrexial and another subnormal, this probably corresponding to outbreaks of blood destruction: the more marked the metabolism, the greater the tissue changes, as in enteric fever, for example. With this there is no emaciation; fat is rather increased; the spleen is not enlarged; but eventually dropsy and serous effusions take place. The patient also often complains of pain and tenderness in the long bones.

The disease is further characterised by its chronic progress, death occurring within two years. The patient dies from blood starvation, the final stage being marked by panting, sighing, waving of arms, as in death from hæmorrhage.

Treatment.—Prolonged rest in bed is most important. We must give a free, nutritious diet, and administer blood tonics. Arsenic seems more serviceable than iron, and may be gradually increased until mxxx . of Fowler's solution can be tolerated at a dose. Under this treatment the disease has occasionally been arrested in its progressive course.

Transfusion appears to be our only further hope.

Chlorosis

Definition.—Presumably a form of anæmia, common in young unmarried women, producing a greenish-yellow complexion, attended by constipation, palpitation, and functional heart murmurs.

Causation—*Sex and Age.*—Young women, between puberty and twenty-five. *Occupation.*—Dressmakers, shop-assistants, domestic servants, and dwellers in underground apartments, or occupiers of badly-ventilated rooms. *Locality.*—More especially a disease of towns, but by no means entirely so. It occurs in country places where the surroundings are unhealthy. *Condition of Life.*—It is far more frequent in working classes than in those well-to-do, and in single than in married women. *Habits.*—It is always associated with constipation and dyspepsia, and probably these two factors are potent in the production of the disease.

Symptoms.—The patient usually complains of breathlessness on exertion, and dyspeptic troubles. Further inquiry reveals that she is suffering from constipation, and amenorrhœa or leucorrhœa. She has a greenish-yellow tint of complexion, and pallor of gums and conjunctivæ. On auscultation there is heard a soft systolic murmur (louder in the recumbent position—A. E. Garrod) over the pulmonary artery, with accentuation of the second sound; there are venous hums of various intensities and characters at the root of the neck; but there is no cardiac hypertrophy. Her feet and ankles are often œdematous. She is not emaciated, but, on the other hand, is generally plump and well-nourished. Frontal headaches and pains in both flanks are common, and she is generally listless and disinclined to exercise.

One or two symptoms require further notice. We cannot overlook the fact that such cases occur almost entirely in young unmarried women, at a period dating from the beginning of sexual activity; further, if chlorosis occur in a married woman we have frequently noted that she has not borne a child. The amenorrhœa is a sequel to the disease, and not a cause. But there is considerable range in the degree of catamenial irregularity; the flow may be scanty, it may be colourless, and it is believed that in many cases it is entirely absent. Cessation, however, probably never occurs; it will be found that in chlorotics there is actually a slight monthly 'show,' some attempt of Nature, however feeble, to carry out her obligations. The urine is abundant and pale.

The pain in the flanks is not inflammatory, but is due to gaseous accumulation in hepatic and splenic flexures of colon.

The dyspeptic troubles are important. One patient frequently has pain and distress after the lightest food, which are only relieved by vomiting. Another patient, although having a depraved appetite, and an aversion for healthy food, has a marked craving for acids and sour fruits. She will eat for dinner a little animal food accompanied by a huge allowance of pickles, or she has a craving for sour apples, unripe fruit, lemon juice, or the like. This class of case is by far the most frequent.

Constipation is almost a constant symptom ; and the late Sir Andrew Clark has ably argued that chlorosis is mainly due to the toxic effects of fecal accumulation, a copræmia, in fact. Those who have had great experience will admit that there is much truth on his side.

When chlorosis has existed some months, the patient frequently returns still chlorotic, but, in addition, with symptoms of gastric ulcer, viz. pain after food, vomiting, which relieves the pain, and occasionally hæmatemesis. Such cases cannot have been gastric ulcer from the beginning, with chlorosis as a consequence, else there would have been vomiting, hæmatemesis, or melæna from the very first. We must conclude, therefore, that there is a pre-ulcerative stage of gastric ulcer attended by all the symptoms of chlorosis, or that many cases of chlorosis which are neglected will eventually develop into ulcer. (See Gastric Ulcer.)

Microscopic examination of blood shows very little or no diminution in the number of red cells ; they, however, have a less tendency than usual to form rouleaux, and contain a diminished quantity of hæmoglobin.

Diagnosis.—The murmur which attends chlorosis is soft and blowing in character, most frequently systolic in period, and loudest over the base of the heart. There is an absence of cardiac hypertrophy or dilatation. Anæmia accompanied by these signs, occurring in a young woman with no previous history of rheumatic fever, is conclusive.

Treatment.—Iron acts as a specific, but it is comparatively useless until the dyspeptic condition is relieved.

The bowels should be first effectually relieved, by castor oil, sulphate of magnesia, aloes, or other aperient ; and it is a good plan to supplement purgatives by large enemata of soap and water with one ounce of olive oil added, and a subsequent daily relief must be insisted on. Meanwhile, relieve dyspepsia by alkalies, with dilute hydrocyanic acid, bismuth, or nux vomica.¹ When the tongue is clean, then begin the iron tonic. The alkaline preparations are best tolerated, the acid tincture of

¹ R. Ext: Cascara: Sagrad: Liq: mx.; Acid: Hydrocyanic: Dil: miiij; Sodæ Bicarb: gr. xv.; Infus: Gentiane Comp: ad ʒj. Misc.

the perchloride of iron often causing pain. Bland's pill three times a day is an admirable preparation, but not better than the *Mist. Ferri Co.* (Griffiths' mixture). The citrate and the tartrate of iron are less astringent than other preparations. We may in certain cases give reduced iron (gr. x. t.d.) or the saccharated carbonate of iron (gr. v. t.d.). An excellent ferruginous water is the Flitwick chalybeate natural water.

Arsenic at times works wonders when iron has failed. It cannot be tolerated when there is any suspicion of gastric ulcer.

Whether iron or arsenic be selected, it must be given for three months at least, as relapses are apt to occur.

It will be found at the onset of treatment that rest in bed for a week or more is of great advantage. The dietary also is important. Food should be given in small quantities at short intervals. Avoid cheese, pastry, pickles, and every article of diet which is in the least degree difficult of assimilation. If stimulant be required a little mild ale may be given, or, better still, a glass of Australian Burgundy with the principal meal.

Leucocythæmia

Definition.—A progressive disease characterised by marked and permanent increase of the white corpuscles of the blood, attended by, perhaps due to, enlargement of the spleen.

Causation.—*Age and Sex.*—Most frequently in males between twenty and fifty—the bread-winners, in fact—who are more exposed to *malaria*, *syphilis*, *fevers*, and all diseases which tend to produce hypertrophy of spleen. If it occur in women it is associated with pregnancy, or the climacteric period, menorrhagia, syphilis, and those depressing influences which appear to be factors in producing grave disorders of the spleen and liver. *Ague.*—Quite 25 per cent. of cases afford a history of paludal exposure; and this is probably underestimated. Patients frequently come from localities which were previously malarial; and, although they do not always bring a history of ague, a mild or 'dumb' attack may have been overlooked.

Pathology.—The splenic hypertrophy mostly affects the

pulpy tissue, the stroma and trabeculae not being so much involved. At times the Malpighian tufts are in a state of amyloid degeneration. Local patches of peritonitis may cause its adhesion to neighbouring organs or abdominal walls, especially in cases where the enlargement has been rapid. The marrow of the long bones is pale, thin, and filled with leucocytes. Hypertrophic changes in the lymph glands and lymphoid tissues are found in the tonsils, the base of the tongue, the glands of the groins, axillae, and in the retroperitoneal glands. In some cases the splenic hypertrophy predominates ; in others, the principal lesions are found in the lymphatic glands, or in the marrow of the bones. The liver is enlarged at the expense of the lymphoid tissue surrounding the portal system of vessels. The heart muscle is fatty and pallid.

Symptoms.—The spleen is enlarged early. It may extend as far downwards as the iliac crest, and transversely to or across the median line. It is detected as a hard, smooth tumour, with its long axis extending from the left axillary line downwards and forwards in the abdomen, its anterior edge being sharp, with well-defined notch or notches. The most remarkable change occurs in the blood. The white corpuscles increase in number till they average 1 in 20 of the red, or even in advanced cases 1 in 3 (normal proportion 1 to 550). They also increase in size, but their nuclei are small. In colour the blood is pale, it contains an excess of fat, its iron constituents are much diminished, and its power of coagulation is materially lessened. Hence, hæmorrhages are common, and may issue from nose, stomach, bowels ; or the catamenia may be excessive and continuous ; in addition, there may be difficulty in staunching the wound caused by slight surgical operation (teeth extraction, &c.), or, rarely, there may be loss of vision from retinal hæmorrhage. The gums become swollen and spongy, the pulse is feeble and intermittent, and there is sooner or later general hypertrophy of the lymphoid tissue throughout the body. As a result of the blood changes, the prominent symptoms are anæmia, with dyspnoea, palpitation, languor, and tinnitus. The temperature is either continuously febrile, or subject to variations ranging from a

subnormal level to 101° or 102° . Although the anæmia and exhaustion are gradually progressive, the fatal termination of the case often supervenes with alarming suddenness.

Diagnosis.—When the spleen is palpably enlarged, the diagnosis is quite easy. In the early stages we must be principally guided by the microscopical evidence of increased number of the white blood-cells.

Prognosis.—Always a fatal disease. Its duration is about two years, but life may be prolonged by treatment.

Treatment.—Remove the patient if possible from a locality having any suspicion of palustral taint, past or present. Even without any malarial history a change of air often works great benefit; a bracing inland health resort, such as Tunbridge Wells or the Scotch Highlands, is to be preferred to relaxing seaside places. As regards medicine, arsenic is our best hope. Begin with miv. of Fowler's solution, and gradually increase the dose until mxx. can be tolerated. At times it may with advantage be taken in combination with iron, as the arseniate of iron ($\text{gr. } \frac{1}{12}$), or with the vinum ferri. Valuable adjuncts to our treatment are the Woodhall Spa and the Kreutznach waters.

Surgeons have proposed and practised extirpation of the spleen. The results do not warrant our advising this step without weighty consideration. It is, however, useless to perform so capital an operation when the disease is so far advanced that the patient has not sufficient vitality to survive.

PLUMBISM

Definition.—The poisonous effects produced on the system by the action of lead salts. The disease is usually a chronic one.

Causation.—The absorption of any preparation of lead into the system. It may be introduced: (i) by the mouth; by water, ale, cider, wines, taken from leaden pipes or vessels; by food when impregnated with lead from unwashed hands. It is believed that the carbonate of lead is decomposed

by the hydrochloric acid of the stomach, and thus absorbed ; hence those subjects who have an excess of free acid appear more prone to the affection. (ii) By the lungs, when it is inhaled as a fine dust in some of the manufactures in which lead carbonate is used, such as card-glazing, making of Brussels lace, &c. (iii) By the skin, from the use of lead-lotions, cosmetiques ; and in certain occupations, as plumbers, compositors, and others who handle lead in any form.

Pathology.—Lead may be detected in all the tissues of the body, and in all the organs and secretions. A form of muco-enteritis is also found in those who have suffered from colic.

The muscles are wasted, especially those which have been paralysed ; and in extreme cases they have undergone fatty degeneration, as a result of their inactivity. There would also appear to be paralysis of the involuntary muscular fibres of the intestines, producing irregular contractions, or even intussusceptions. As yet there is some doubt as to the primary lesion caused by the poison. By some observers (Henle) the poison is thought to attack the involuntary muscles ; by others it is held that it attacks the nervous system, either at the periphery, or at the anterior horn of the grey matter in the spinal cord.

Symptoms.—There is no regular sequence of symptoms. The first indication may be colic, or muscular paralysis, or profound anæmia. For convenience only, the symptoms may be grouped into—

(1) **AFFECTIONS OF THE ALIMENTARY CANAL.**—A blue line is found in the gums round the neck of the teeth, due to the deposit of lead sulphide ; the gums are retracted ; there is a metallic breath ; also nausea, occasional vomiting, marked constipation, and at times tenesmus. With these there is often colic, occurring in paroxysms, the pain being of a tearing character, chiefly referred to the umbilicus, worse at night, and relieved by pressure. The abdominal walls at the same time are hardened and retracted.

(2) **NERVOUS.**—Paralysis, first of the extensors of the forearm (*extensor communis digitorum* is the first muscle

involved); thence it extends to other muscles of the arms, trunk, and legs. The extensor ossis metacarpi pollicis and the flexors usually escape, the supinator longus being no exception to the rule, and so do the interossei and lumbricales; ¹ hence we find 'dropped wrist' or a stooping and shuffling gait. The palsied muscles show R. D. Ultimately the affected muscles cease to respond to galvanic currents even, and the deep reflexes are absent. In advanced cases there may be convulsions, delirium, or insanity. Cases have also been recorded with blindness, due to atrophy of the optic nerve.

(3) VASCULAR SYSTEM.—Marked dingy anæmia and cachexia, with diminution both in the number of red corpuscles and in the quantity of hæmoglobin. Gout is frequent, as the presence of lead in the system tends to arrest the excretion of uric acid.

(4) GENITO-URINARY SYSTEM.—Polyuria is a notable feature, the urine being albuminous, of low specific gravity, and presenting other signs of chronic Bright's disease. Abortion occurs in pregnant women.

Diagnosis.—The dropped wrist is almost characteristic of chronic lead poisoning. But colic, together with the presence of blue line in the gums, the pallid and dejected appearance, can only point to lead as the cause.

Prognosis.—Favourable if treated before the disease is far advanced. Severe lesions of the eyes and kidneys are of course permanent.

Treatment.—(a) PROPHYLACTIC.—The patient should either give up his occupation if it brings him in contact with lead, or practise scrupulous cleanliness by washing his hands, brushing his teeth, changing clothes, using respirators, &c. Food should never be eaten in the workshop where lead is employed. The lead workers should be encouraged to drink lemon-water to which dilute sulphuric acid is added.

(b) CURATIVE.—First relieve the pain. Colic may be temporarily relieved by the application of hot fomentations

¹ It is a noteworthy anatomical fact that the flexors and adductors are respectively and collectively stronger than the extensors and abductors.

or poultices to the belly. The next indication is to procure an action of the bowels. Calomel or castor oil may be given internally in combination with opium.¹ The best results, however, are obtained by administration of sulphate of magnesia, to which sulphuric acid is added.² The free acid is supposed to form an insoluble salt of lead, which is purged from the system by the magnesia salt. Iodide of potassium (grs. x. to xv.) has also been advocated with a view to the elimination of lead by the kidneys. A long-continued course is necessary. We have generally seen good effects, however, by the administration of large enemata of soap and water with olive oil, followed by the ordinary mist. alba in ounce-and-a-half doses until the bowels act. The palsied muscles should be treated by electricity. Apply a slowly-interrupted or constant feeble current daily, the sittings lasting ten minutes. Beyond this we may obtain help from iron tonics when there is much anæmia and strychnia to help to restore tone to the affected muscles.

MERCURIALISM

Definition.—The poisonous effects produced on the system by the action of mercury. The condition may be acute or chronic.

Acute Mercurialism

Symptoms.—Mainly those of an irritant poison. After a poisonous dose we should find colic, cramp in the belly, diarrhœa with mucous stools, suppression of urine. The tongue is white and shining ; there is a metallic taste in the mouth ; the skin is clammy ; the pulse feeble ; and death occurs from collapse, being often preceded by muscular twitchings and convulsions.

Treatment.—Albumin in some form or other should be administered at once. Give eggs beaten up in milk or flour

¹ ℞. Hydrarg : Subchlor : gr. iij. ; Pulv : Opii gr. j. Ft. pulv.

² ℞. Magnes : Sulph : ʒj. ; Acid : Sulph : Dil : mx. ; Aquæ Chloroformi ad ʒj.

mixed with water to the consistency of cream. Allay thirst by milk, and lime water.

Chronic Mercurialism

Causation.—It is chiefly observed amongst workers at trades in which crude mercury or the oxide is used. It therefore affects those engaged in the manufacture of mirrors, barometers, &c. It has also been known to supervene on a long course of mercurial treatment, whether by the mouth, or by inunction, or by the use of mercurial lotions. Some subjects are particularly prone to be early affected. The poison (the oxide especially) may enter the system by the respiratory passages, by the mouth (food), or by the skin. Crude mercury itself may be given in large doses without poisonous symptoms.

Pathology.—Postmortem examination would reveal a general muco-enteritis, but especially in the rectum ; together with, it may be, sloughing and ulceration of the mucous membrane. The kidneys are inflamed and the urinary bladder is contracted. Careful analysis detects the presence of mercury in most of the tissues.

Symptoms.—(1) GASTRO-INTESTINAL.—Salivation is, as a rule, the first sign, accompanied by a silvery, glazed tongue and a characteristic fœtor of breath. The teeth become brittle and exfoliate ; there is often a gangrenous ulceration of gums and buccal cavities ; and a red translucent line at the edge of the gums, has been observed. With these there are diarrhœa and tenesmus, the stools being of a mucous character. (2) NERVOUS.—There are characteristic tremors of muscles involving first those of the upper extremities, and gradually extending to the trunk and lower extremities. The gait thus becomes ‘dancing’ and irregular. The tongue is tremulous, and the speech jerky, as in disseminated sclerosis. These symptoms are followed by failure of memory, mental obfuscation, or even mania, and ultimately death, preceded by delirium. (3) RENAL.—The urine is scanty or almost suppressed, and may contain blood.

Diagnosis.—FROM PARALYSIS AGITANS.—This disease oc-

curs usually in late life. The muscular tremors are present when the patient is at rest ; they seldom involve the tongue, and one would expect to find the characteristic gait (festination).

FROM DISSEMINATED SCLEROSIS.—This disease is distinguished by the presence of nystagmus, the scanning speech, and the increase of deep reflexes.

FROM ALCOHOLISM.—By the history of drink, the alcoholic odour, and absence of salivation.

Treatment.—**PROPHYLACTIC.**—If possible, remove the patient from contact with mercury. Enjoin strict cleanliness both as regards food, clothes, and person. **CURATIVE.**—Give albumin freely ; eggs, bread, flour, and milk are easily available, the albumin in these substances forming an insoluble compound with mercurial salts. Iron filings have also been given with success. Treat the buccal symptoms with gargles of alum or of common salt. Opium is indicated in severe abdominal pains. Recovery will be facilitated by vegetable tonics combined with iodides.

ACUTE ALCOHOLISM (DELIRIUM TREMENS)

Definition.—A disease due to acute alcoholic poisoning, characterised by delirium, sleeplessness, and muscular tremors.

Causation.—(a) **PREDISPOSING.**—*Age and Sex.*—Men between twenty and fifty are most frequently affected. Alcoholic women, although they occasionally suffer from delirium, appear, from their tendency to secret drinking, to develop peripheral neuritis and other manifestations of chronic alcoholism. *Condition of Life.*—Starvation, exposure, and mental depression. *Occupation.*—Publicans, brewers' labourers, and others whose occupations (or whose want of occupation) lead them to excessive alcoholic consumption. But cases occur in all ranks and callings. *Injuries and Febrile Diseases.*—Especially severe fractures, lacerations, crushings ; also pneumonia, specific fevers, &c. ; but there is invariably a history of previous alcoholic transgression.

(b) **EXCITING.**—Alcohol, in some form or other. It may be produced by the purest wines or spirits, but is more especially due to abuse of impure and adulterated alcohol. Most

frequently there is a history of a sudden debauch supervening on habitual excess.

Pathology.—The principal pathological changes are found in the gastro-intestinal canal. The mucous membrane of the stomach is inflamed (gastritis), and a similar condition occurs in the small intestines (enteritis). The extent of the lesion varies from acute congestion to intense inflammation, with here and there extravasation and hæmorrhages. The cortex of the brain is usually deeply engorged, and a similar condition affects the medulla. Serous effusions may be found in the pericardium and other cavities. Alcohol can be discovered by chemical analysis in all the organs and tissues, notably in the brain, liver, and intestines.

Symptoms.—There are three diagnostic symptoms, viz. : sleeplessness, tremors, and suspicions. These may appear at once, and with no premonitory signs, during a drinking bout ; but usually there is a history of an ante-delirium state, characterised by loathing of food, restlessness, and disturbed sleep or absolute wakefulness. Subsequently, and somewhat abruptly, the patient becomes delirious. He does not sleep, or at least sleeps badly. He has alarms ; he sees animals, monsters, and devils ; or he fancies he is being pursued, or is about to be captured and tortured. He is suspicious of his best friends. He has all manner of delusions and of hallucinations affecting the senses of sight, hearing, smelling, and taste. Every article of furniture may be a lurking place for some devil or fearful object, or it may represent to him something totally different, whether painful or pleasant. Generally his fantasies are of a distressful character ; but occasionally they are the reverse, and he imagines his room is strewn with gold, or filled with beautiful women, or the walls hung with splendid pictures, and the like. His fancies often vanish during the daytime, to return in the same or in some altered form at night. Or his delirium may be related with his occupation (‘ busy ’ delirium), and he must needs, according to his calling, get out of bed to draw ale for customers, or to attend some exalted patient, or to address the senate, or to preach to a crowded congregation. With all this aberration

of intellect he can usually be recalled temporarily to his senses ; he is amenable especially to his doctor, or to someone who has moral control over him. We have often suspected that this may, in no small degree, be due to a tone of authority of voice to which he is not continuously accustomed.

His muscular system also presents marked signs ; he suffers from tremors, especially on movement ; he has minute twitchings of tendons (noticeable in feeling his pulse) which may amount to marked subsultus. His face and conjunctivæ are congested, and his expression is dull and vacant. The tongue is foul and tremulous, his breath offensive, and his bowels are irregular. Usually there is a history of diarrhœa prior to his delirium ; the bowels and other secretions then become locked up on cessation of stimulants, looseness returning when alcohol is again taken.¹ The pulse is soft and frequent, the rate being 120 beats. His temperature is not in accordance with the amount of delirium. It is increased, but it does not, as a rule, except in severe unfavourable cases, exceed 100° F.

If rigid abstinence from alcohol be enforced, and food taken, the patient may sink into sleep, from which he wakes coherent and much improved ; but, on the other hand, in spite of treatment his symptoms may get worse. His mental condition then is more pronounced ; he may become maniacal or suicidal ; his pulse increases in frequency and becomes 'running' ; his tongue gets brown and dry ; his temperature increases (103°-4-5°) ; his muscular tremors are more marked ; subsultus and carphology are present ; and ultimately the patient sinks in coma.

Diagnosis.—(1) FROM INSANITY.—Here the onset is gradual, the delusions are more persistent, the muscular tremors are usually less pronounced, and there is probably no history of alcoholic excess.

(2) FROM MENINGITIS.—In this disease the violence is greater, there is intolerance of light and sound, also more prostration and tendency to coma ; nor are these symptoms cured by sleep. In addition, the pulse is wiry and intermittent, and

¹ A reversed condition is observed in the morphia habit.

the tongue is not foul, nor is it tremulous. Should meningitis occur in a dipsomaniac, the diagnosis is very difficult ; but we should ascertain previous habits.

(3) FROM HYSTERIA.—Here the patient is more obstinate ; there is muscular rigidity rather than tremor ; there is no rise in temperature or in pulse ; finally, a cure does not always result from food and sleep.

(4) FROM TYPHUS FEVER.—By the absence of petechial rash, and of the characteristic mousey odour ; and by the history of alcohol.

Prognosis.—Good, as a rule. Few patients die in their earlier attacks. The prognosis is more favourable if sleep be procured and food taken. The more unfavourable signs are a high and increasing temperature, and a feeble dicrotic pulse. Few pronounced alcoholics entirely give up their evil habits, and therefore attacks are liable to recur, each succeeding one rendering the prognosis more unfavourable.

Treatment.—There are three main indications, viz :—

(1) *Entire Abstinence from Alcohol.*—There is, in the absence of cardiac disease, no danger in taking liquor completely away from the patient. He is suffering from alcohol, so why continue it ? It is idle and waste of time to diminish the quantity gradually. Nor are there any good grounds for believing that the delirium is due to total abstinence in one who was previously a toper. It is also advisable to administer a purgative, so as to get rid of any alcohol from the intestinal canal.

(2) *Get the Patient to take Food.*—It is best administered in a liquid form, and should consist of milk (three pints a day) ; two eggs beaten up with the milk or cooked together as a custard ; beef-tea or beef-essences in quantity equivalent to half a pound of flesh. If these forms be refused we may resort to highly-peppered soups or broths, jellies, &c. ; or, lastly, to nutrient enemata. But food must be given in some form or other.

(3) *Procure Sleep.*—The patient should be in a quiet, secluded, and shaded room ; he should see no friends ; and he should have, if possible, one attendant only, or one for day-

time and another at night. Restraint is not advisable unless he be very violent. It is as well, however, to remove all knives and dangerous implements, and also to secure the windows and doors. There is much dispute as to the necessity of soporifics, but there can be none as to the value of sleep. Our own experience is decidedly in favour of such remedies, and especially opium. If it be decided to give opium, let it be administered at once, in full doses, and repeated if necessary; the indications for opium, as a rule, outweigh those against it. It may be given in one-grain doses, either in the form of pill, or the tincture (℥xv.), every three hours until sleep is secured; some prefer a subcutaneous injection of morphia (gr. $\frac{1}{2}$). We may give fuller doses at night and smaller ones through the day. If there be albuminuria, and for that reason opiates be discarded we may order chloral (grs. x. to grs. xxx.) or bromide of ammonium (grs. xxx.), but the latter does not, as a rule, act speedily enough. Excellent results follow the administration of digitalis (℥x. to ℥xxx., or more) every four hours, especially in cases where the kidneys are injured and the pulse is feeble and dicrotous. Some advocate tincture of cannabis indica (℥xx.), which may be combined with bromides; but its effects must be watched with care; in excess it produces symptoms not unlike delirium tremens itself. After a sleep of about twelve hours the patient is practically cured; but he still requires care and rest.

Gastric symptoms, such as nausea and vomiting, may be relieved by small doses of tincture of nux vomica (℥iij.) with ipecacuanha wine (℥ij.). Chloride of ammonium is indicated in active congestion of the liver. Subsequent craving for alcohol is relieved by tincture of capsicum with some bitter infusion, or by oxide of zinc, or the mineral acids.¹ After recovery the patient should, if possible, be removed from his injurious surroundings and taken to the sea-coast or to some bracing inland resort. Tonics (quinine and iron) will be required in addition.

¹ R. Acid: Nitro-Hydrochlor: Dil: ℥x.; Tinct: Capsici ℥x.; Spt Chloroformi ℥xv.; Infus: Chiretta ad ℥j. Misco.

CHRONIC ALCOHOLISM

Definition.—A condition due to the habitual consumption of an excess of alcohol, which produces symptoms pointing to disease, both functional and organic, of the nervous, digestive, and other systems.

Causation.—(a) **PREDISPOSING.**—*Age.*—From early adult age to end of life. It is more frequently met with, however, between thirty and forty years of age than later, probably owing to the fact that alcoholics are, as a rule, short-lived. *Sex.*—Males especially, but both sexes predisposed. *Heredity* is often strongly marked. *Habit and Occupation.*—It may commence in carelessness, the habitual use of a stimulant, ‘nipping,’ business transacted ‘over a glass’; it is, therefore, prevalent amongst commercial travellers, brewers’ employés, publicans, cattle-dealers, and others. *Mental Emotion*, whether of a depressing or an exciting character, such as grief, despondency, anxiety, joy, success. *Insanity.*—The probability seems that in many cases a mental weakness existed from the first. When the habit is once contracted it is astonishing to see how the victim is a complete slave to it. It, to all purposes, is a form of insanity; and it is not an infrequent incident for a dipsomaniac, when told by competent authorities that his propensity will cost him his life or his position, to order or procure more liquor as soon as his friends have left him.

(b) **EXCITING.**—Alcohol. In large towns spirits (whisky, gin, brandy, &c.) are the prevalent poison. In country districts ale is the predominant drink. Among the affluent, wines and spirits appear to be consumed indiscriminately.

Symptoms.—No subject has a more important bearing on the previous medical history of a patient, with the exception of syphilis and, perhaps, acute rheumatism. The quantity of alcohol consumed and the length of the chronicity vary in different individuals. Some appear able to take daily for many years, and without any apparent injury, an amount of alcohol sufficient to permanently injure three people at least. Nor is

there any order or priority of symptoms. In one the chief disturbance appears in the stomach, in another in the liver, whilst a third may show failure of nervous system. We have therefore classified the chief symptoms according to the various systems.

(1) GASTRO-INTESTINAL CANAL.—The breath is foul: at times offensively sweet, somewhat as in diabetes; in others it has a hot, bilious odour; the tongue is coated and ragged, or abnormally clean and red; the throat and fauces are congested; there is a disgust for meals, especially breakfast; morning sickness is common, even before there are physical signs of liver disease; the bowels are irregular, constipation alternating with bilious diarrhœa. The liver is generally enlarged and tender in the early stages; catarrhal jaundice is not uncommon; whilst in advanced cases a bronzed jaundice, observed in advanced cirrhosis, may be present.

(2) URINARY SYSTEM.—The kidneys are not so often affected structurally as one would suppose; occasionally there is albuminuria, with other signs of chronic epithelial changes (large, pale kidney); but far more frequently there is glycosuria, with a low specific gravity.

(3) CUTANEOUS SYSTEM.—The face is blotchy from acne; the capillaries of the nose, chin, and cheeks are engorged; the conjunctivæ are injected; and there is a bleary-eyed, heavy, and listless expression. Further, the patient is liable to boils, carbuncles, and sores on different parts of the limbs and trunk.

(4) CIRCULATORY SYSTEM.—The heart is often irritable, and occasionally gives signs of fatty changes; the pulse is permanently accelerated; valvular disease, especially aortic regurgitation, is a frequent lesion.

(5) RESPIRATORY SYSTEM.—Congestions of bronchial tubes and of the bases of the lungs are common; and in the latter stages pneumonia may occur as a complication.

(6) NERVOUS SYSTEM.—The gait is shuffling and ataxic; the patient complains of want of, or disturbed, sleep; his memory is deficient or obfuscated; and he may suffer from epileptic attacks, or even dementia. Changes in his moral character

are, however, most marked. He is not truthful ; he is cunning, or dishonest ; he has a want of determination and fixity of purpose ; he is often cowardly and boastful ; and when at his lowest depths will associate with people much below his position, and will, when in poverty, consume even spirituous tinctures, eau de Cologne, or any liquor containing alcohol. Nevertheless, he clings to life ; he hopes for improved health ; and appears joyous and delighted if his medical adviser can only find the least improvement in his symptoms.

(7) MUSCULAR SYSTEM.—The voluntary muscles are wasted (want of nutrition), especially in the limbs ; yet his abdomen increases in size, and his figure, which once may have been spruce and athletic, becomes bloated and frog-like. Subsequently there is general wasting of muscular and adipose tissues throughout the body ; and he dies from the effects produced by the alcohol on the liver (cirrhosis) or on the brain (hæmorrhage), or from phthisis, or from fatty changes in the heart.

Treatment.—The only preventive is total abstinence. Even after restraint under the 'Habitual Drunkards' Act, and apparent cure, it is absolutely dangerous to allow the minutest quantity of alcohol in any form. If alcoholism be a form of insanity, as appears most probable, the craving produced by the slightest transgression after abstinence may cause a return to the old debauched condition. The craving may be ameliorated by *nux vomica*, the mineral acids, capsicum, phosphorus ; but even at best these drugs are impotent unless supplemented by determination on the part of the patient and watchful care and sympathy by his friends. A change of air, scenery, and surroundings are useful. Beyond these, good wholesome food is essential, in order to repair the tissue waste and damage.

DISEASES OF THE DUCTLESS GLANDS

ANATOMY

The **Spleen** weighs about seven ounces in an adult. It is situated in the left hypochondrium, and, except in children, or when enlarged, it cannot be felt below the costal arch. Its external surface, smooth and convex, is in relation with the diaphragm, which separates it from the ninth, tenth, and eleventh ribs. The internal surface is concave, and presents a vertical fissure, or hilum, the edges of which give attachment to the gastro-splenic omentum.

In front of the hilum the spleen is in relation with the cardiac end of the stomach ; below, it comes in contact with the tail of the pancreas ; and behind with the left suprarenal body.

The upper end is rounded, and suspended to the diaphragm by a fold of peritoneum. The lower end is pointed, and rests on the end of the transverse colon. The anterior border is sharp, and presents a well-defined notch. The posterior border is rounded, and rests on the left kidney.

Its blood-supply is from the splenic artery, a branch of the cœliac axis. The artery is tortuous, probably to accommodate itself to altered sizes and positions of the organ. The splenic vein joins with the superior mesenteric to form the portal vein.

The **Thyroid Gland** weighs from one to two ounces. It is larger in females, especially during menstruation. It is situated in the neck, on the trachea, and consists of two lateral lobes joined by an isthmus which runs across the second and

third rings of the trachea. Each lobe reaches from about the fourth ring of the trachea to about the middle of the thyroid cartilage, but this extent is by no means uniform. It is overlapped on each side by the sterno-hyoid, sterno-thyroid, and omo-hyoid muscles. The edge of each lobe rests on the carotid sheath and its contents. A third middle lobe occasionally exists.

The arterial supply is suggestive of the importance of the gland, since it receives a superior thyroid branch from the external carotid, and an inferior thyroid branch from the thyroid axis of the subclavian ; both vessels, as it were, going out of their way to supply the gland.

The **Suprarenal Capsules**, one on each side, rest on the upper end of the right and left kidney respectively, being connected to these organs by areolar tissue. Anteriorly the left capsule is in relation with the spleen and pancreas, the right with the under surface of the liver, to which it is usually found adhering when that organ is removed from the body. They lie on the respective crura of the diaphragm, at a level with the tenth dorsal vertebra. The inner border is in relation with the great splanchnic nerve and the semilunar ganglion, the aorta also being in contact with the left, and the inferior cava with the right capsule. Their separate weights are about one to two drachms.

DISEASES OF THE SPLEEN

Congestion

Causation.—There are two forms : (*a*) **ACTIVE**, resulting from malarial, typhoid, typhus, pyæmia, and other specific fevers ; and also febrile conditions which produce hyperæmia of this organ. (*b*) **PASSIVE**.—A venous engorgement, secondary to disease of the liver, portal obstruction, chronic bronchitis, emphysema, and valvular diseases of the heart.

Pathology.—The spleen is enlarged, firm, very red on section, and easily lacerated. On microscopic examination the vessels are engorged, notably the smaller arteries and veins, and also the sinuses or venous bags, as in erectile tissues.

Occasionally there is rupture of some of the vessels, with extravasation into the splenic tissue (apoplexy), or escape of blood through the serous coat into the peritoneal cavity.

Symptoms.—The spleen is enlarged. Normally, lying on the ninth, tenth, and eleventh ribs, in the left axillary line, it may extend upwards as high as the eighth, but more especially downwards and forwards into the left lumbar and umbilical regions, so that it can easily be felt on palpation. When enlarged by congestion the gland retains its shape ; it is tender ; it can be easily manipulated and moved, and its characteristic notch detected. It also ascends and descends with the movements of the diaphragm.

Acute Inflammation

Causation.—This may result from blows, punctures, and other traumatism. Medically, however, it is usually the

result of infarctions, secondary to fevers, or to valvular disease of the heart.

Pathology.—When the whole organ is inflamed from injury, it is enlarged, intensely red on section, with its capillary vessels engorged. When due to infarcts the inflammation is local and in patches. Infarcts are triangular in shape, with their apices towards the hilus, and their bases towards the convex surface, where they frequently form bulgings of varying sizes under the peritoneal coat. On section, an infarcted area is darker than the surrounding part, being of a purple or plum colour; after a time it becomes pale and colourless from obstruction of the circulation to the part; subsequently the area may soften and break down, forming a small abscess; absorption then takes place, leaving a puckered cicatricial tissue; and, if it be near the surface, adhesion to parietal peritoneum. In fevers and in valve disease the infarcts are, as a rule, not numerous; but in pyæmia several small foci of inflammatory changes may be observed in any given section of the spleen.

Symptoms.—There is enlargement of the spleen attended by febrility. The splenic tumour is painful and tender; it still presents a notched anterior border; and it moves on respiration, unless extensively adherent to the stomach or to the abdominal parietes. The limits of the tumour may vary from a slight encroachment upon the abdominal cavity to an extension as far as the linea alba in front, or to the crest of the ilium below, or backwards as far as the dulness of the erector spinæ muscles.

Treatment.—Apart from the treatment of the primary disease which occasions splenic inflammation, little can be done beyond leeching, or cupping, supplemented by hot fomentations and poultices.

Atrophy

Causation.—Wasting diseases and malnutrition are the most frequent causes. It may also be secondary to chronic inflammation, or the result of thickening of its serous coat after peritonitis.

Symptoms.—None very definite. There is some localised pain, followed by anæmia, and a decrease of the normal dullness of the organ.

Treatment is the same as that of the disease which occasions the disorder.

Hypertrophy

Causation.—It is usually a secondary complication to some blood disorder, such as malaria (most frequently), or Hodgkin's disease, leucocythæmia, rickets, hereditary syphilis, and chronic heart disease.

Symptoms.—In malarial hypertrophy there are usually the symptoms attending prolonged or repeated attacks of ague. The skin is of a characteristic dirty brown tint; the face is wrinkled, and the expression anxious and aged; the blood contains an increased number of white corpuscles, varying in amount according to the extent and chronicity of the disease; there are hæmic murmurs to be heard at the various cardiac orifices, notably over the pulmonic artery, and also over the vessels at the root of the neck; the stomach and bowels are disordered and irregular. On examination of the spleen, that organ will be found greatly enlarged, but of course varying in degree in different cases ('ague cake'); it is firm to the touch, generally painless; its edge retains its sharp outline and characteristic notches. Usually the liver shares in a corresponding hypertrophy. Finally, all subsequent ailments have a tendency to be stamped with a periodicity of exacerbation and remission of symptoms. The disease is usually chronic and endemic in India, but may disappear on removal to a healthier climate. (See also Hodgkin's Disease, Leucocythæmia, Rickets.)

Pathology.—The spleen is enlarged, weighing, it may be, eighteen to twenty pounds avoirdupois (seven ounces normal weight), yet it retains its relative shape. It is firm and hard to the touch, from hyperplasia of its connective tissue, although all its histological elements are increased. Usually the organ has an increase of pigmentation.

Treatment belongs to the disease of which it is a sequel (see Ague, Leucocythæmia, &c.). It may, however, be advisable in certain cases to apply iodine over the enlarged organ, or to apply belladonna and other soothing plasters. In the enlargement due to syphilis, iodide of potassium with mercurials should be given internally.

New Growths

Tubercle.—Usually in cases of acute tuberculosis the spleen suffers in common with the other organs of the body. The tubercular growth may, therefore, be of the miliary type, or, if the patient survive long enough, it appears in the form of aggregated yellow masses, which ultimately caseate or break down, forming irregular, unhealthy-looking abscess cavities.

When the spleen is involved in acute tuberculosis, the organ is enlarged ; it is usually adherent, by inflammation of its surface, to the abdominal walls, as there is generally tubercular infiltration of the peritoneum also.

Malignant New Growth.—Primary cancer of the spleen is very rare, although cases have been recorded. Usually it is a secondary condition ; and when it occurs, it begins at the hilus and extends thence along the vessels and the trabeculae to the interior. Encephaloid is the most frequent form of cancer. Sarcoma may also, at times, involve the organ.

There are no special signs or symptoms until the growth be large enough to cause irregularities in percussion and palpation.

Lardaceous Disease

Definition.—A degenerative change, common to the spleen and other vascular organs of the body, produced by prolonged suppuration.

Causation.—Phthisis, chronic abscesses, caries of bone, and other diseases attended by suppuration. The degeneration is never limited to the spleen.

Pathology.—The organ is symmetrically enlarged, smooth, with its borders rounded and blunt. The notch in the anterior border, therefore, is not so distinct as in simple

hypertrophy. The lardaceous material affects the small arteries generally, and also the Malpighian tufts and the trabeculæ, but not all to the same degree necessarily. The Malpighian tufts being involved, they are seen as pale or greyish lumps, quite distinct from the surrounding tissue, and thus giving rise to the name *sago spleen*. They stain readily with solutions of iodine.

LYMPHADENOMA (HODGKIN'S DISEASE)

Definition.—A disease characterised by profound anæmia, with hypertrophy of the lymphoid tissues throughout the body, accompanied by paroxysmal pyrexia.

Causation.—(a) PREDISPOSING CAUSES.—*Age and Sex.*—A disease especially of children, or early adolescence, principally affecting boys. *Condition.*—It occurs oftenest amongst the poor, and those who can only procure deficient and improper food. *Prolonged Irritation* of skin, mucous membranes, or any tissue in intimate relation with the lymphatic system. It may thus have its starting-point in decayed teeth, chronic inflammation of nose or pharynx or middle ear, or in one of the chronic suppurative diseases of the scalp.

Pathology.—It bears a strong resemblance to malignant neoplasm, especially to the sarcomata, inasmuch as it appears to be engendered by irritation, at a period of life when the vascular and lymph organs appear to be in a state of developing activity.

The glands which are affected may be, at first, those draining the vicinity of some sore or irritation, and the enlargement is local only; but after a time induration of other lymph glands occurs in scattered localities; even in those sites where glands are agminated, it is only one or two in each bunch which become involved at first.

The infection then spreads to neighbours until they form knotted masses, the component parts of which appear welded together so as to form lobulated tumours. In the earlier stages the glandular increase appears to consist mainly of the cellular elements; at later dates the fibrous tissue is principally con-

cerned in the hyperplasia, so that the glands consist of fibro-nucleated stroma ; they eventually become homogeneous, not unlike the condition seen in amyloid degeneration. When the spleen is enlarged, as it often is, it presents scattered masses, yellowish in colour, of lymphoid tissue, which, as a rule, form trabeculæ or bands, slightly raised above the cut surface, but which may in transverse sections appear as rounded or globular masses. A similar hypertrophy may appear in the liver, kidneys, and intestines, and indeed in most parts of the body, owing to the well-known general distribution of lymphoid tissue. The softer form of glandular enlargement, that condition when cells of an embryonic type are proliferated, is more 'malignant' and more rapid in its fatal course than when the glands are firm and hard from completed structure. The marrow of bones may be pale, thin, and filled with white blood-cells, as in leucocythæmia.

Symptoms supervene gradually. Enlargement of lymph glands may be first noticed, those of the neck being usually first involved ; but similar enlargements may occur in the glands of the axillæ, the groins, popliteal spaces, or mediastinum. On the other hand, anæmia may first attract our attention. As a rule, the anæmia increases with the enlargement of the glands, until the pallor is very extreme. The white blood-cells are relatively increased, though not to such a degree as in leucocythæmia. They are smaller in size than in leucocythæmia, but their nuclei are larger. Beyond these two signs, the one distinctive feature is a tendency to paroxysmal pyrexia. The temperature may suddenly rise from a little above normal to 102° or even to 103·5° F. without any obvious alteration in the glandular tumours, except perhaps increased tenderness, and without any disease of respiratory, circulatory, or other organs which we can detect by our methods of exploration. The spleen is, as a rule, enlarged, though nothing like as in leucocythæmia ; but splenic enlargement is not necessary ; indeed, the disease may exist without it. The liver also is usually hypertrophied, and perhaps painful. There is in addition a tendency to hæmorrhages from all organs and into all cavities. Death usually is

due to the disorder of the blood ; but it may occur from pressure of the new growth on some vital organ, or from obstruction of important artery or canal (trachea, œsophagus, &c.). The disease in its clinical features resembles 'malignant' new growth, and is by some, perhaps, rightly considered as such ; but by its microscopical distribution the new growth is distinctly homologous, being merely an excessive outgrowth of lymphoid tissue, and should therefore be regarded as 'innocent.'

Diagnosis.—FROM LEUCOCYTHÆMIA.—The two diseases resemble one another in many symptoms. In both there is an excess of leucocytes in the blood, but this is much more pronounced in leucocythæmia. In the latter disease also, splenic hypertrophy is an early and predominant sign ; in Hodgkin's disease the lymphatic glands are first affected, and the spleen enlarges subsequently only.

FROM SCROFULA.—In this disease the lymphatic hypertrophy is not so general, and the glands are not matted. We should also obtain evidence of tubercular deposit in the lungs or other organs.

Treatment.—Remedy any diseased condition of mucous membranes of buccal cavities, such as hypertrophied tonsils, pharyngeal adenoids, carious teeth, &c. The iodides of potassium, sodium, and ammonium exert important alterative action on all mucous surfaces. Large doses are not necessary, two to three grains of the iodide of potassium three times a day are ample.

For the glandular enlargement arsenic is a valuable remedy ; it should be given in cautiously increased doses, as in leucocythæmia ; or we may inject a solution of arsenic directly into the different groups of glands, with good effect. Surgery promises great help, however. We have seen large colonics of glands extirpated from beneath the sterno-mastoid and trapezius muscles with marked success. An indurated gland is a source of danger, and if it does not respond quickly to medicinal treatment the sooner it is eradicated the better. Cod-liver oil and iron are powerful adjuncts ; and we may also order prolonged residence at the seaside of some of our more bracing eastern and northern resorts.

MYXŒDEMA

Definition.—A disease of adult life, due to destructive change of the thyroid gland, characterised by a general mucus-yielding œdema, accompanied by slowness of movement and also of mental processes. The nomenclature is Dr. Ord's.

Causation.—(*a*) **PREDISPOSING CAUSES.**—*Age.*—Most cases occur between thirty and sixty-five, but others have been recorded at all periods. *Sex.*—Females, in the proportion of six to one.

There is an absence of evidence connecting the disease with heredity, occupation, social status, or family history. As regards the last point, the disease has not been recognised sufficiently long for us to form any definite conclusion.

(*b*) **EXCITING CAUSE.**—Atrophy, destruction, or removal of the thyroid body. This appears to have been conclusively proved by surgical operations (Kocher), by vivisectional research (Horsley), and by pathological examination (Ord).

Pathology.—Centres on the thyroid body. This organ is involved in a 'destructive change,' which 'consists in the substitution of a delicate fibrous tissue for the proper glandular substance. A similar interstitial development of fibrous tissue is also observed . . . in the skin, and with much less frequency in the viscera, the appearances . . . being suggestive of an irritative inflammatory process.' Eventually the gland is completely atrophied and replaced by fibrous tissue.

Chemical examination of the various tissues generally has revealed the excess of mucin, especially in the skin.

Symptoms.—The first recognised symptom is the œdema of the skin of the body generally, but earliest recognised in the face. It produces, therefore, a characteristic 'facies.' The features are broad, puffy, and coarse; the eyelids are pale and transparent; the nostrils broadened; the lower lip thickened and everted; and the mouth widened with its curves obliterated. Usually there is a well-defined blush on the cheeks; the skin is cold, dry, and scaly, especially on the chest and on the extensor surfaces of forearms and hands; there is an absence of perspiration and sebaceous secretion, hence the skin is harsh

and wrinkled ; the hands are broad, clumsy, and expressionless ; the nails are brittle ; the hair is stunted, ragged, and readily falls out. This symptom is first shown in the scalp : the parting of the hair gets widened and scaly, and eventually there is almost complete baldness. Similarly, the teeth fall out and the gums recede. Frequently there have been observed supraclavicular lumps or hypertrophies of the fatty, or vascular, or connective tissues of this part. Cutaneous sensibility is blunted, the differentiation of sensations of heat and cold being retarded. Usually the patient feels cold, and her bodily temperature is actually subnormal. She complains more of these symptoms in winter than in summer. Her muscular activity is similarly affected : the gait is ponderous, the movements of the hands and of the body are slowed ; thus she is a long time in dressing or in doing her household work, and she herself is conscious of this apparent lethargy. In addition, there is usually some muscular paresis ; the head often falls on the chest, or she drops things which she may have been holding ; occasionally there is actual rupture of muscular fibres, owing to a want of co-ordination. As regards the digestive system, the tongue is foul and swollen ; an œdematous thickening is observed in the palate and uvula ; speech is therefore deliberate and the voice 'leathery' in intonation. The appetite does not appear to be affected, but the bowels are generally constipated. The urine has a diminished specific gravity, contains a lessened quantity of urea, but does *not* contain albumin, or at least not until the latest stages.

In the vascular system the heart and blood-vessels may be said to be normal ; at any rate, there are no constant marked signs. But when the disease is well-established, hæmorrhages are not uncommon and are difficult to arrest. Hence there is some risk attending teeth extraction, or other surgical operations, also in parturition and in injuries and bruises of all sorts. The menstrual flow may also be excessive.

Nervous symptoms are important, and, as a rule, well-defined. Not only is the patient slow in speech, from œdema of the soft palate, as stated above, but she is slow in

thought ; she fails in her memory, and, as a reflection of this, her speech is halting and deliberate : she is equally slow and painstaking in writing. On the other hand, she may be loquacious and emotional. As regards temperament, the sufferers are usually patient and placid, and they sleep well ; but this calm is often interrupted by outbursts of temper, and rudeness or suspicion. Delusions and hallucinations also are not uncommon. In addition, they may complain of headaches, of rheumatic pains in the joints, and of disagreeable tastes or smells, and other subjective symptoms. On careful digital examination for the thyroid in a thin subject, it will be found small and almost absent ; but estimation of its size is so difficult in an ordinary subject that we must rely on the general symptoms narrated above. (See 'Clinical Soc. Trans.,' supplement to vol. xxi.)

Diagnosis.—FROM CHRONIC BRIGHT'S DISEASE.—There is an absence of albuminuria in myxœdema. The expression of face also, and the condition of the skin and of the scalp complete a picture which really has no resemblance to any other disease.

Treatment.—Till recently no remedy had been regarded as in any way arresting the disease. The patient should be well and warmly clad, and should, if possible, remove during winter to some warm and sunny place. Her general health should be improved by tonics, careful diet, and gentle exercise in the open air ; and we must direct our treatment to the amelioration of distressing or dangerous symptoms as they arise.

Dr. Ord has had good results from the use of jaborandi (tincture $\text{m} \times$. t.d.), or the nitrate of pilocarpin (gr. $\frac{1}{32}$ to gr. $\frac{1}{2}$). We also have seen remarkable relief from it in one case under our care. Its chief effect, from the statement of patients, is in promoting the action of skin, and rendering them warm and comfortable. In view of the pathology, however, Horsley suggested that transplantation 'of a thyroid from a healthy sheep to a myxœdematous patient might arrest the disease,' and the experiment was tried with success. Then Dr. G. R. Murray injected the juice of a sheep's thyroid with great benefit.

Subsequently Dr. Hector Mackenzie boldly gave a patient two sheep's thyroids to be eaten, with the result that there was 'a general diminution of the swelling and amelioration of all the symptoms.' (See 'B. M. J.' Oct. 29, 1892). Mackenzie, from observations made in two cases, recommends half a thyroid every day. In three cases under our own care we found no difficulty in giving the thyroid when finely minced and placed between bread and butter as a sandwich. We gave half a gland twice a week, and in four weeks the results were certainly marvellous.

GOÎTRE

Definition.—Enlargement of the thyroid gland, due to hypertrophy of one or more of its histological elements.

Causation.—*Age and Sex.*—A great preponderance of cases occurs in females between ten and twenty. It is comparatively rare in men, and seldom commences in either sex after middle age. *Locality.*—It prevails endemically in the Swiss valleys, in Derbyshire, and other parts of the Midlands where the potable water is derived from the limestone formation. But, singularly, it does not appear frequently on the South Downs, where the water is strongly impregnated with chalk. It appears in some way to be associated with the commencement of puberty, and with pregnancy. It is doubtless propagated by intermarriage of close relations. Cases were more commonly met with in Derbyshire before railways opened out the country; and statistics show that it is less prevalent than formerly in Swiss valleys which are now made accessible. It is interesting to observe (and we have seen a number of examples) that frequently the children of goitrous parents are cretinoid, or at least imbecile.

Pathology.—Chiefly, if not entirely, in relation with the thyroid gland, which becomes hypertrophied. The hypertrophy may vary from a slight fulness of throat to a huge pendulous tumour the size of a child's head. Nor is the enlargement always symmetrical; one side may be much more involved than the other. Microscopically there are four varieties of hyper-

plasia, viz. : (1) VASCULAR, in which the arteries, and especially the veins, are enlarged and engorged, the gland then being soft and pulsating. (2) FIBROUS, in which there is an increased growth of the connective tissue of the organ—a cirrhosis, in fact—so that the tumour is hard and firm. This variety frequently undergoes calcareous change. (3) CYSTIC, in which the loculi of the organ are greatly distended with fluid or blood, or a semi-gelatinous substance, probably an altered condition of the normal fluid filling the vesicles. This condition forms a tense, fluctuating, globular tumour ; its shape, however, will vary according to the distribution and size of the loculi. (4) A MIXTURE OF THE PRECEDING CONDITIONS.—From some microscopic preparations we have been led to believe that a goître may commence with one pathological change (say the cystic) most pronounced, and that after a time this remains in abeyance, and possibly the activity of the hypertrophy may be transferred to the vascular or to the fibrous tissue elements. All the various forms of goître are liable to inflammation and its results, including hæmorrhages and suppuration.

Symptoms.—Mainly dependent on the discomfort and weight of the tumour and the pressure it exerts. As a rule, the enlargement is painless, if unsightly, and beyond this there may be no trouble, even in large growths ; life is easily tolerated, and even heavy manual work may be carried on. But it may exert pressure on neighbouring structures, and give rise to some of the signs of aneurysm. Thus, the trachea may be compressed antero-posteriorly or laterally, or it may be dislocated to one side ; the œsophagus may be squeezed (dysphagia), especially if the left lobe of the thyroid be greatly involved ; or there may be abductor paralysis of vocal cords from pressure on the recurrent laryngeal nerves. Should the goître extend to the upper opening of the thorax, its pressure may involve venous trunks, causing localised œdema ; or affect the lower trachea and bronchi, giving rise to cough, stridor, coarse rhonchi, and the like. Other symptoms occasionally associated with goître are mental depression or irritability.

Diagnosis.—Presents no difficulty. The tumour is usually bilateral, and moves up and down with the larynx.

Treatment.—Iodine, by common consent, appears to be the best remedy. It may be given internally (tincture, or as iodide of potassium); it may be applied externally; or injected directly into the tumour. This requires some care and discretion, as suppuration, with high fever, may follow.

Others recommend mercurial inunctions (hydrarg. biniodid. or hydrarg. oleat.), followed by exposure to the sun or fire. The mercurial inunction has had good effects in our hands; but we doubt the advantage of exposure. We sometimes, in addition, try the effects of fluoric acid (m℥. to ʒj.) ; of iodide of sodium (grs. iv. to grs. x.) ; of phosphorus (gr. $\frac{1}{30}$ in pill). Blisters, frequently repeated, occasionally relieve by diminishing the size of the growth. We may puncture cysts and inject them with diluted tincture of iron; finally, we may recommend total extirpation of the growth, or division of the isthmus. Caution the patient beforehand of the tendency to myxœdema which follows this operation. At any period of the disease pressure signs on the air-passage may become suddenly acute, and may therefore demand tracheotomy.

ADDISON'S DISEASE

Definition.—A disease, due to destructive changes in the supra-renal bodies, characterised by vomiting, progressive debility and anæmia, together with marked pigmentation of the skin.

Causation.—(a) **PREDISPOSING.**—*Age.*—No period of life is exempt, but it occurs most frequently in middle age. *Sex.*—Men are affected oftener than women. *Mental anxiety* and *Depression* are supposed to be causal factors.

(b) **EXCITING.**—Tubercular disease of the supra-renal bodies; but it is probable that any destructive lesion of these organs may directly cause the disease.

Pathology.—The essential lesion is situated in the supra-renal bodies. These organs are in the early stages enlarged,

and adhere to surrounding structures. When examined microscopically they are found to be infiltrated with a finely fibrillated connective tissue, containing inflammatory corpuscles in its meshes. At this stage the capsule is translucent and tough. Subsequently tubercular masses, containing tubercle-bacilli, develop; these ultimately caseate and break down, so that the interior of the organ becomes converted into a pulpy, cheesy, or creamy substance, as in the degenerative changes of yellow tubercular deposits elsewhere. Beyond these lesions of the capsules, the inflammatory process extends to the neighbouring nerves, and the connective tissue in which they are embedded. The cerebro-spinal (vagi and phrenics), and also the sympathetic nerves with their ganglia (solar plexus and semilunar ganglia), are involved, so that a lesion occurs which closely resembles the condition seen in sclerosis.

It is yet an undecided point whether any pathological change in the adrenals, other than tubercle, will cause the symptoms of Addison's disease. Evidence, however, tends to support the view that malignant new growth, or even atrophy alone, of these bodies will cause symptoms which are indistinguishable from those caused by tubercular deposit.

As regards the skin and some of the mucous membranes, there is a marked increase of pigmentation in the deepest cells of the epidermis. But it is not limited entirely to these cells. A thin line of pigmentation in scattered granules is found in the true skin. This line is quite distinct from, and runs parallel to, that of the more superficial discoloration, and it follows the various elevations and depressions of the cutis.

Symptoms.—There is no priority of symptoms, but the typical discoloration of skin may be preceded by a period marked by intense debility and prostration, pains in the loins, and occasional gastric disturbances (vomiting). Sooner or later the characteristic pigmentation of skin becomes apparent. The discoloration is not patchy, though some parts are darker than others; nor is it a true bronzing, as there is no metallic lustre, but merely a browning of the affected skin. It is most marked on the backs of the arms, folds of axillæ, in the

groins, the scrotum, and the nipples, also round the waist or round the knees where belts and garters exert pressure. The colour is not limited to the skin, but may also involve sub-mucous tissues of the tongue and buccal cavity, and also the labia minora; in these last localities the pigmentation is decidedly patchy. As the disease progresses, the heart's action becomes feeble; there is dyspnoea; the gastric disturbances increase; the patient complains of chilliness, especially in the lower extremities; there is usually an absence of fever, indeed the temperature may be subnormal. The flame of life may be regarded as slowly burning out.

In the final stage the skin and breath have a characteristic corpse-like odour; the patient becomes dull and apathetic, or delirious, and he finally sinks from exhaustion.

Diagnosis.—**OCCUPATION.**—The darkening of the skin may be simulated by a similar condition which occurs in asphalters, and gas-workers. Tramps and vagabonds (vagabondismus) also present a discoloration of skin, which at times may resemble Addison's disease. **PREGNANT WOMEN** also show pigmentation in various parts of the skin. **NITRATE OF SILVER.**—A prolonged course of this drug may produce permanent staining of the skin. The diagnosis of Addison's disease, however, does not rest on pigmentation alone; there must be in addition marked symptoms of debility, together with wasting, vomiting, and nervous symptoms. Note the complexion of the patient: should his hair be light and his skin dark coloured, it would be suspicious in combination with any of the above symptoms.

Prognosis.—A fatal disease; death usually occurs within a year of the commencement of marked symptoms. Life may be prolonged by judicious nursing and diet. Recently we have had under our care a patient, with remains of discoloration still evident, who was under care in Guy's Hospital twelve years ago, where the case was diagnosed as Addison's disease. And in view of the possibility of arrest of tubercular disease elsewhere, it is probable that such may have happened in this case. It is also conceivable that a portion of the adrenals may have remained unaffected, and so carried on the functions of these organs.

Treatment.—As the disease is a progressive one, we can only hope to prolong life by rest, warmth, and nourishing food. Cod-liver oil with tonics (arsenic or iron) appear to give best results. We must relieve vomiting and other symptoms as they arise. Dyspeptic trouble may be relieved by alkalies, with dilute hydrocyanic acid. Stimulants may be given when required.

DISEASES OF THE URINARY SYSTEM

ANATOMY

The **Kidneys** are situated in the loins. They extend from the level of the upper border of the twelfth dorsal to the level of the middle of the third lumbar vertebra ; or from the eleventh rib to an inch above the highest point of the iliac crest, the right kidney being a little the lower of the two.

Each organ rests on the crus and lower part of the diaphragm, the psoas, and the anterior lamella of the transversalis aponeurosis, which separates it from the quadratus lumborum.

Anteriorly the relations differ somewhat on the two sides. They are overlapped by—

<i>The Right</i>	<i>The Left</i>
Right lobe of liver	Tail of pancreas
Second part of duodenum	The stomach
Ascending colon	Descending colon
Peritoneum	Peritoneum

Of each kidney the upper end is somewhat enlarged and rounded, forming a kind of head. The lower end is more pointed.

The inner border is situated about one inch external to the vertebral spinous processes.

The outer border is about three and a half inches from the same 'landmark.'

The anterior surface is convex. The posterior surface is more flattened. The vessels at the hilum are, from before

backwards, vein, artery, and ureter. The artery is highest, the ureter lowest. The weight of each organ is from four to five ounces, the two about equalling the weight of the heart.

The **Ureter**, starting from the hilum, runs almost vertically downwards, to enter the bladder obliquely in the posterior false ligament, below the obliterated hypogastric artery, the vas deferens passing between it and the bladder. It is entirely covered by peritoneum.

In the female it reaches the bladder by passing by the side of the cervix uteri or upper part of the vagina.

On each side it rests on the psoas and the common iliac artery at about its bifurcation. Anteriorly it is overlapped by the spermatic vessels and ileum on the right side, and by the spermatic vessels and sigmoid flexure on the left side.

The length of the tube is about seventeen inches. Its course would be represented by a line drawn on the anterior abdominal wall from a point two inches external to and above the umbilicus, down to the spine of the pubes.

The **Bladder** is a pelvic viscus when empty, but rises into the abdomen when distended.

In children it is conical-shaped, and situated higher up than in adults. Its anterior surface, uncovered by peritoneum, is in relation with the posterior layer of the triangular ligament, the back of the pubes, and, when distended, with the anterior abdominal wall.

The posterior surface, covered with peritoneum as far forward as the obliterated hypogastric arteries, is in relation with the rectum in the male, and the uterus in the female, some coils of small intestine intervening. The base in the male rests upon the second portion of the rectum, from which it is separated by the recto-vesical fascia. This portion of the bladder corresponds to the triangular space of which practically the apex is at the prostate, the sides formed by the vesiculæ seminales and vasa deferentia, and the base by the reflected recto-vesical fold of peritoneum. This fold may extend almost to the prostate if the bladder be empty.

In the female the base of the bladder is in relation with the cervix uteri, and is attached to the anterior wall of the vagina.

Peritoneum separates the bladder from the anterior surface of the body of the uterus.

The axis of the bladder would be represented by a line drawn from the coccyx to the anterior wall of the abdomen at some point between the umbilicus and the pubes, the point varying according to the distension of the viscus.

THE URINE

In Health.—Urine should be straw-coloured, clear, of acid reaction, of specific gravity between 1015 and 1025 ; and it should deposit, after standing, a small cloud of mucus. The quantity passed *per diem* in health amounts to about fifty-three ounces. It should contain neither albumin nor sugar.

It may, in disease, vary from these normal characteristics.

Transparency.—It may be turbid, from decomposition, from excess of mucus, or from the presence of pus, phosphates, blood, semen, and, in rare cases, chyle. Turbidity after cooling is due to the precipitation of amorphous urates. A urine which is extra clear and transparent is often unhealthy ; this is especially seen in diabetes and contracted granular kidney.

Colour.—May vary from a very pale yellow tint to brownish-black. Extreme paleness with high specific gravity generally denotes the presence of sugar ; with normal specific gravity it is significant of anæmia. A reddish mahogany tint is often associated with liver disorders and indigestion. In acute Bright's disease the urine is 'portery' or smoky, whilst the prolonged use of carbolic acid, creasote, turpentine, either internally or externally, often produces a brownish olive or even black colour. Other drugs, such as santonin, gamboge, turn the urine yellow, whilst rhubarb and senna produce a reddish tint. As a general rule, it may be stated that highly coloured urines are of high specific gravity, and pale urines the reverse.

Reaction.—May vary between extreme acidity in gouty urine, and the marked alkalinity caused by decomposition. This decomposition is entirely due to micro-organisms which

break up the urea and lead to the formation of carbonate of ammonia. Urine is also slightly alkaline after food or alkaline medicines, which convert the soluble acid into insoluble alkaline phosphates.

If the alkaline reaction be due to ammonia, the urine will turn a red litmus slip blue, this colour, however, being lost by warmth; whereas, if the alkalinity is due to the fixed alkalies (potash or soda), the blue tint is permanent.

The normal acidity due to presence of urates and acid sodium phosphate may also be increased by acid medicines, by opium (Harrison), by excess of animal food, and in febrile states generally.

Quantity.—The extremes would be represented by almost total suppression in cholera on the one hand, and polyuria amounting to fifty pints *per diem* in diabetes insipidus on the other. We must, however, distinguish between suppression and retention. It is also necessary to note the influence of season of year, and of sex. The condition known as Hydronephrosis may seriously modify the quantity from day to day, at one time the excretion being much diminished, at another increased.

The urine is generally diminished in all febrile diseases, in intestinal obstruction, in acute Bright's disease, after violent perspiration, vomiting, or diarrhoea, and by limitation of the amount of fluid consumed.

It is increased (polyuria) in chronic Bright's disease, in lardaceous disease of kidneys, in diabetes, and in hysterical or neurotic subjects; also in convalescence from acute disease, during the absorption of serous accumulations, and by the action of drugs, such as scopolarium, juniper, digitalis, and potash salts.

The **Specific Gravity** ranges in health from 1015 to 1025. It shows the relative amount of solid constituents present in the urine; a high specific gravity generally indicating the presence of sugar or an excess of urates, whilst a low specific gravity is found in hysterical polyuria, and in some forms of Bright's disease. In estimating the solid matter in a sample of urine, multiply the last two figures of the specific gravity by 2·3. The result will give the amount of solid matters per

1000. For example, a urine is of specific gravity 1025 : then $25 \times 2.3 = 57$ per 1000 (about). The daily amount of solids passed can of course only be estimated by examining a specimen from the whole of the urine passed in twenty-four hours.

Mucus to a certain amount is always deposited in healthy urine, and it may be greatly increased in catarrhal affections of the bladder.

In Disease.—*Abnormal constituents of urine* may be divided into: (A) those which are ordinarily in solution, and (B) those which are deposited (Ord).

(A) The principal constituents held in solution are, *albumin, sugar, bile, colouring matter, urea in excess.*

Albumin.—There may be a faint trace in contracting kidney, or from the presence of pus or (menstrual) blood. There may be such an amount as to render the urine solid on boiling, as in acute nephritis.

It is, however, very frequently observed in a minor degree in children under ten years of age, and is probably of little significance. If constant, however, it indicates disease of the kidneys or urinary apparatus.

The causes of albuminuria are classified into: (1) LOCAL ; (2) GENERAL.

(1) LOCAL CAUSES.—Inflammation of kidneys, bladder, or any part of urinary tract ; vaginal discharges in women ; injuries to kidneys or bladder ; renal calculi ; pressure on renal veins by tumours (new growth, pregnant uterus, &c.) ; thrombosis of renal veins ; or embolism of renal artery.

(2) GENERAL CAUSES.—It may be due to venous engorgement, secondary to chronic heart or lung disease ; it occurs in acute febrile disorders, either during the attack, as in diphtheria and typhus, or as a sequel of scarlet fever and erysipelas. Occasionally it is a complication of nervous disorders, such as cerebral hæmorrhage, due to degeneration of the arteries, which is common in advanced renal disease ; or it may be consequent on some new growth of the nervous system, such as a tubercular mass, or a syphilitic tumour. It supervenes also on general blood disorders, as in lardaceous degeneration, leucocythæmia, purpura, also on malignant disease,

syphilis, phthisis, hæmorrhagic diathesis (bleeders), and pregnancy. Albuminuria may also be induced by errors of diet, or by failure of digestion. It is then often temporary only, occurring especially after meals.

Finally, it may be due to the influence of drugs, such as turpentine, cantharides, mercury, lead, and others.

In all cases of albuminuria a record of the specific gravity and a microscopic examination of the deposit are of the utmost importance. (For chemistry, see p. 526).

Sugar in the urine to any marked degree, if associated with polyuria, a high specific gravity, and with thirst, may be taken as confirmatory of the existence of diabetes mellitus.

It may, however, occur in small, yet appreciable quantity in cases of cerebral disease; in patients over fifty who habitually eat and drink too much (hepatic congestion); in cases of angina pectoris, gout, and in tertiary syphilis. It has been observed after suppressed lactation. Minute traces are also frequently found in healthy urine. (For chemistry, see p. 527.)

Bile never occurs in healthy urine. When present, it is significant of jaundice, present or oncoming. The urine is thus stained according to the amount of bile present, and varies from an orange tint to a dark bottle-green. Both bile acids and bile pigment may be present. (For chemistry, see p. 529.)

As regards other colouring matters, the chief are *Purpurin*, *Indigo*, and *Uro hæmatoporphyrin*.

Purpurin—a product of febrile disorders and diseases of liver—is easily detected, when present in any quantity, by its staining urates and other crystalline deposits.

Indican—a product of indigo-blue and indiglucon—is present in many abnormal states. It is a colourless body which by excess of strong acids is converted into indigo-blue and indigored. From repeated observations made by Dr. Ord and myself we concluded that indican is present in many disorders of the peritoneum and gastro-intestinal tract. Notes of cases show that it is most frequently (almost invariably) present in tubercular peritonitis; but it is also easily discovered in cases of obstinate vomiting, gastric ulcer, enteric fever, dysentery,

malignant disease of bowel or peritoneum, diarrhoea, and intestinal catarrh. In all cases children's urine appears to give a better reaction than that of adults. (For Chemistry, see p. 530).

Urohæmatoporphyrin—a derivative of hæmatin—has been found by A. E. Garrod in acute rheumatism.

Urea being very soluble is deposited only if in great excess. Normally it forms about two per cent. of the urine, 512 grains *per diem* being excreted. It may be free or, most frequently, combined. It is observed in excess in gout, diabetes, febrile disorders, and derangement of the hepatic digestive function. It is, however, as a diminished urinary product that the examination for urea is most important. The amount daily excreted is lessened in all diseases in which the liver-secreting structure is destroyed (acute atrophy, cirrhosis, &c.) ; in all forms of Bright's disease ; or as a result of starvation. (For chemistry, see p. 530.)

(B) The principal abnormal constituents which are deposited may be subdivided into : (1) CRYSTALLINE ; (2) NON-CRYSTALLINE.

(1) Of CRYSTALLINE deposits the chief are amorphous urates, phosphates, oxalate of lime, uric acid, cystine, leucin, tyrosin, and carbonate of lime.

(a) *Amorphous urates* of soda and ammonia appear as a dust or mud, which may be pale or coloured according to tint of urine. They are dissolved by boiling, and are re-deposited on cooling ; they commonly stain the vessel in which the urine is contained. Urates are generally indicative of sluggish hepatic action, overfeeding (animal food especially) and abnormal muscular exertion. They combine with acetic and other acids to form uric acid. In combination with soda they are often passed by children in the form of 'hedgehog' crystals. The amorphous urate, however, does not follow any definite crystalline formation.

(b) *Phosphates* occur in two varieties, the ammonio-magnesian phosphate (commonest) and the phosphate of lime. The triple, or ammonio-magnesian, phosphate appears usually in prisms ('knife-rests,' or 'house-tops'), occasionally in feathery crystals. They are deposited in ammoniacal,

decomposing urine, and are usually associated with vesical catarrh. They may, however, denote the presence of a phosphatic calculus. Phosphate of lime, seen usually as radiating rods or stars, is said to occur in wasting diseases and in nervous prostration. (For chemistry, see p. 531.)

(c) *Oxalates of lime* appear as fine powder or dust, and are highly refractive. Although found in small amount in healthy urine, if in large and persistent quantity they suggest the presence of calculus; generally, however, they denote some form of dyspepsia. They are frequently found after eating rhubarb, gooseberries, and other products of the vegetable kingdom. The urine which contains them is acid or neutral. Their usual form is octahedral ('envelopes'); but in albuminous urine they often fall as 'dumb-bells,' owing to the presence and influence of colloid matter. (See Ord, 'Influence of Colloids upon Crystalline Form and Cohesion.') (For chemistry, see p. 531.)

(d) *Uric Acid* is deposited in reddish crystalline particles resembling cayenne pepper (sand or gravel); colourless themselves, they are usually stained yellow by the urinary pigment. They are found in acid urine, and when pure are insoluble in water, but are readily attacked by nitric acid and by liquor potassæ. Their forms are various, the most usual being barrel- or lozenge-shaped crystals; they have also been likened to double-edged chisels. They frequently occur as superimposed tablets or rosettes, and occasionally as stars or spikes. The crystals are particularly large, and well-defined if the urine contains sugar (Ord). A 'dumb-bell' form of crystal is extremely rare, except in albuminous urine. When habitually present in any quantity, uric acid may be diagnostic of gout, Bright's disease, hepatic disturbance, indigestion, and calculus. (For chemistry, see p. 531.)

(e) *Cystine*.—A rare product of urine, deposited in hexagonal, and often superimposed, laminae. It contains about twenty-six per cent. of sulphur. The urine has an oily appearance and a sweet odour. The conditions giving rise to cystinuria are not known. It is said to 'run in families.' We have seen cystine passed in great quantities by a patient

during an attack of pneumonia, and also in a case of hypochondriasis, the cystinuria being probably the cause of the nervous condition. (For chemistry, see p. 532.)

(*f*) *Leucin* and *Tyrosin* occur in the urine of patients suffering from acute atrophy of liver, typhus and typhoid fevers. Leucin is deposited as oily-looking discs, and occasionally as plates. Tyrosin crystallises as fine needles, or as acicular, radiating, or feathery-looking masses. Their clinical significance is at present little understood.

(*g*) *Carbonate of lime* occurs in small spheres or minute calculi. It is a rare product of human urine; and beyond the suggestion of a calculus, it has probably little clinical significance.

(2) NON-CRYSTALLINE.—The chief are, blood, pus, fat, chyle, casts of renal tubes, epithelium, cancer cells, spermatozoa, parasites, bacteria, and sarcina. We must also bear in mind introduced foreign bodies, such as faecal matter, starch, hair, &c.

Blood colours urine in various shades, according to the origin and quantity of the hæmorrhage. Thus, urine may present the slight smokiness due to the intrusion of blood from catamenial flow, or it may be of such a dark red colour as to be scarcely distinguishable from pure blood.

As regards the source of hæmorrhage, it may come from kidney or from urinary tract below (ureter, bladder, prostate, or urethra). If it come from the kidney the urine is uniformly mixed with blood and is smoky, a grumous deposit falling after a time. If from the bladder or prostate the urine is pink in colour, the blood is often found in clots, which generally appear at the end of the stream; whilst if the hæmorrhage come from the urethra, clots are generally expelled with the urine first voided. All these general statements, however, must be received with modifications in certain cases.

Renal hæmorrhage is chiefly caused by injury, calculus, new growth, embolism or thrombosis of renal vessels, parasites, inflammations, and abscess. Certain poisons also appear to exert a topical effect, viz.: cantharides, turpentine, and others.

If the hæmorrhage be from bladder or prostate, the conditions giving rise to it are mainly surgical, and would include injury, calculus, new growth, parasites, inflammation, and also prostatic hypertrophy or ulceration. Symptoms such as pain at the neck of bladder and in the hypogastrium, together with frequent desire to micturate, would point to the bladder as the seat of the hæmorrhage.

The causes of urethral hæmorrhage are likewise chiefly surgical, and may be enumerated only. They would include injury and stricture, chancre, gonorrhœa, calculus, and occasionally new growth.

On the other hand, hæmaturia may be quite unconnected with lesion in the urinary apparatus, but may be symptomatic of some general disease. It occurs in hæmatinuria, purpura, scurvy, specific fevers (small-pox, typhus, scarlet fever), and in hæmophilia. In such cases the blood is freely diffused through the urine, producing a uniform 'smoky' or 'portery' tint.

The diagnosis of the various causes will depend on many collateral symptoms, which can only be discussed in the descriptions of the various disorders named above. The microscopical evidence, however, as a diagnostic element is essential. (For tests, see p. 532.)

Pus may come from any part of the urinary tract, but especially from the kidney or bladder. In quantity it may vary from the minute trace revealed only by the microscope to such an amount as to constitute nearly half the bulk of the specimen. As a symptom of renal inflammation, the pus after settling, has a clear, sharply-defined margin separating it from urine above, which is generally acid in reaction, or at least neutral. It may be caused by nephric or perinephric abscess, renal embolism, tubercle, parasites, or by calculus, either in the substance of the kidney or its pelvis. Occasionally seen in Bright's disease, it would therefore contain renal epithelium.

In pus from the bladder the urine is usually alkaline from decomposition, and has, in addition, much superimposed mucus, thus giving, after settling, a graduated line where it merges with the urine above. Here it is symptomatic of

vesical catarrh, calculi, or new growth. Abscesses in connection with uterus, rectum, prostate, or neighbouring parts may also discharge themselves by the bladder. It may also occur in cases of gonorrhœa or of vaginal inflammations. (For tests, see p. 533.)

Fat, apart from chyle and foreign intrusion, is a rare constituent of urine. It is present occasionally in cases of phosphorus poisoning, and in fatty degenerative changes of kidney. Its presence in the urine after catheterism should always be remembered.

Chyle is found in the urine of patients suffering from invasion of the parasite *Filaria sanguinis hominis*. The urine is milky in appearance; it also coagulates spontaneously, owing to the presence of fibrin in addition to fat; this latter subsequently rises to the top of the vessel, like cream. The fatty character of chyle may be demonstrated by its solubility in ether.

Casts are albuminoid mouldings formed in the renal tubules. They consequently vary in calibre according to the diameter of tubule in which they are formed. Their length seldom exceeds one twentieth of an inch. They all consist of a coagulated albuminous substance as a basis, and may have blood-cells or epithelium adherent to their sides.

Those most commonly met with in urinary deposits are : *Hyaline* ; *Blood* ; *Epithelial* ; *Granular* ; *Fatty* ; *Mucous* ; and *Lardaceous*.

Hyaline casts are generally significant of chronic renal disease. They are large transparent cylinders, highly refractive, and have a tendency to be transversely fissured. They are readily stained by colouring matters.

Blood casts indicate acute disease. They have an albuminoid basis in which compressed blood-cells are embedded. Their colour is yellowish-brown. Free blood-cells are nearly always found in addition to blood-casts.

Epithelial casts usually denote acute renal disease; the cells of which they are formed, therefore, may consist of the recently shed epithelium of the tubules mixed with migrant leucocytes.

The epithelial cells vary in shape and outline according to varieties of tubule from which they are shed, and also according to changes to which the epithelia have previously been subjected.

Granular casts are generally significant of chronic or sub-acute inflammation of the kidneys. They are opaque cylinders of varying sizes, studded with finely granular debris or degenerating epithelial cells. Their borders are irregular and ill-defined, and their ends rounded. They would appear to be epithelial casts, in which the epithelium is undergoing degenerative changes.

Fatty casts are indicative of chronic and fatty changes in the renal epithelium. They may present themselves as waxy forms studded with highly refractive oil-globules, or as epithelial casts in which the cellular elements are filled with oil as a result of fatty degenerative changes.

Mucous casts are probably not true casts, but are shreds of epithelium and of basement membrane. They are found in urethral, prostatic, and vesical irritation, and beyond being an aid to diagnosis are probably of little or no significance. They are ill-defined, thin, and often spirally twisted bodies, sometimes of great length, but to a careless observer difficult to detect.

Lardaceous casts are usually large cylinders somewhat resembling hyaline casts. They stain readily by iodine, and are assumed to be mouldings of lardaceous matter which has escaped into the renal tubules.

The clinical significance of casts cannot be overestimated. By careful examination of them we have a direct index of the state of the renal secretory surfaces. It is necessary, however, to judge as to the pathological condition of the kidney, not by the existence of a single cast of a special variety, but by the preponderance of certain forms, since a urine may hold specimens of all. Our diagnosis and prognosis must, therefore, be based on the greatest frequency of any variety or varieties contained in several specimens of the same urine. The situation of the moulding of these casts is a matter of some dispute; but remembering the greater calibre of the proximal convolutions

above, as compared with the loops of Henle below, it is obvious that they cannot have been exuded in the former tubes, unless we are to assume that by pressure from behind they can be compressed so as to fit the vessel of narrower lumen.

Epithelium in small quantity is contained in the deposit of every specimen of urine; but unless the quantity be excessive, or shed in masses or plates, it has no clinical significance. Epithelium may fall from any part of the genito-urinary tract, the exact site of the irritation, therefore, being determined by the different distinctive characters of the epithelial cells. Thus—

From the kidney they are cubical or angular or cylindrical.

- | | | | |
|---|----------------------|----------|---------------------------|
| „ | pelvis | „ | flattened. |
| „ | ureter | „ | pear-shaped or irregular. |
| „ | bladder | „ | ‘transitional.’ |
| „ | prostate and urethra | they are | columnar or pear-shaped. |
| „ | vagina | they are | large, squamous. |

If the epithelial shedding be profuse it is indicative of local catarrh. The cells, therefore, may present no deviation from normal type; or they may have granular or fatty contents, according to intensity or chronicity of the inflammation. Cancer cells are not unlike the epithelium of certain parts of the tract. A profusion of irregularly shaped cells would be suspicious of new growth, but we cannot depend on their number and microscopical appearances alone in our diagnosis.

Spermatozoa.—Of no clinical significance, unless in excessive and repeated quantities. Their presence in urine is generally indicative of prostatic irritation. Discovery of them by the microscope is easily made owing to their characteristic ‘kite’ shape.

Parasites.—Those most frequently met with are, the ova of bilharzia hæmatobia, filaria sanguinis hominis, and small hydatid cysts or shreds of membrane. They give rise to varying symptoms pointing to irritation of the genito-urinary tract, of which any part may be their habitat. The most frequent sign is hæmaturia. Diagnosis is mostly or entirely made by microscopical examination of the urinary deposit for ova, hooklets, &c.

Bacteria and Sarcina.—Micro-organisms bear a most important part in the causation of urinary decomposition, since urine will keep fresh for an indefinite time if carefully excluded from the air. The action of these bodies is greatly increased by an irritable condition of mucous membrane. Vibriones are found in urine undergoing decomposition. Bacteria may also be found in perfectly fresh urine, in which case the excretion is turbid when voided, and continues so. Such conditions are generally due to catarrh of bladder, or follow on catheterism. In females it may be associated with vaginal discharges.

Sarcinæ in urine are somewhat rare.

Mould and torula cerevisiæ are not infrequent in acid urine.

Intruded foreign bodies may be observed, such as hair, dust, wool, and feathers from bedding, &c. Starch ('powder-puff') is often found in women's urine.

The *odour* of urine may also lead to the detection of disease. It is ammoniacal and offensive after putrefactive changes; it has a sweet smell in diabetes; turpentine produces a scent which has been compared to violets.

URINE TESTS

Albumin (serum).—(a) **BOILING** a sample of urine, in the presence of an acid in excess, produces a flocculent precipitate if albumin be present. Acidity is necessary, as albumin will not be precipitated in an alkaline solution. If even the minutest quantity of albumin be present, the urine will 'kick' on boiling. An easy rough estimate of the quantity of albumin may be made by boiling (say) a drachm of urine and then pouring it into a minim graduated glass; the amount of the precipitated albumin can then be easily read off when it has settled down at the bottom of the measure.

(b) **COLD NITRIC ACID.**—Carefully pour pure nitric acid down the inside of a specimen glass into a sample of urine. The two fluids do not readily mingle, owing to their different specific gravities. If the urine contains albumin a cloudy

stratum will appear at the layer of junction of the two fluids. The reaction caused by the formation of crystalline nitrate of urea must not be mistaken for albumin.

(c) **PICRIC ACID** (Johnson's test).—Add equal parts of a saturated aqueous solution of picric acid and of urine in a test-tube and shake. If the urine is albuminous, a well-marked cloudiness will appear, which is made flocculent on subsequent boiling, if desired. The cloudiness is more evident the greater the amount of albumin present. This test is usually the most reliable and convenient of all ; it is easily applied, it can be carried about without injuring other things, and it is very rapid in its action. Further, it can be used as a test for glucose. It can also be applied, after the method of the cold nitric acid test, by filling a pipette with the picric acid solution, and then gently floating it on the upper layer of albuminous urine in a test-tube, urine being, as a rule, of greater density than the picric acid solution. A film will be seen at the junction of the two fluids.

The picric acid test is, however, open to one or two fallacies. Thus, if there is much mucus in the urine, it will produce cloudiness ; but the opacity is 'satiny' in appearance, somewhat refractive, and quite different, therefore, to the dull cloud produced by albumin. It will precipitate an excess of urea, also quinine and other alkaloids which may be passed by the urine. These, however, disappear on subsequent boiling.

Numerous other tests may be used for the detection of albumin, but they are more convenient for laboratory than for clinical work.

Sugar.—(a) **FERMENTATION TEST.**—Take two samples from the whole of the urine passed in twenty-four hours, and place them in separate specimen glasses. To one, add a piece of German yeast the size of an acorn, or some brewer's 'barm ;' then keep both samples in a warm place for twenty-four hours. The mouths of the glasses should be protected by placing a card or a covering of stiff paper over them. On the second day, after complete fermentation has taken place in the one, decant both specimens and take their respective specific

gravities. On comparison, a loss of one degree of specific gravity in the fermented specimen equals one grain of sugar per fluid ounce of urine.

Example :—

1038 = specific gravity of unfermented urine.

1012 = specific gravity of fermented urine.

26 = number of grains of glucose per ounce of urine.

Suppose the patient passes x ounces of urine *per diem*, then $26 \times x = y$, the total amount of sugar passed.

Fermentation is the only reliable test, as uric and hippuric acids both have a slight reducing action in the copper tests. (See Halliburton : 'B. M. J.' vol. xi. 1892.)

(b) FEHLING'S TEST.—This is both a qualitative and a quantitative test, and depends on the reducing power of diabetic sugar on a solution of cupric sulphate of known strength—200 grains of the Fehling's solution are reduced by one grain of sugar.

Take 200 grains of Fehling's solution and boil it in a flask together with an equal portion of distilled water ; then take some of the saccharine urine and also dilute it (1 in 10) with distilled water ; place the diluted urine in a burette graduated to grain measures. Then gradually drop the diluted urine into the boiling Fehling's solution, till all the blue colour vanishes ; the amount of urine required for this represents the quantity which contains one grain of sugar.

Example.—If 1085 grain measures of diluted urine were expended, then $\frac{1085}{10}$ or 108·5 grains of urine contain one grain of sugar.

(c) PAVY'S TEST.—This is a modification of Fehling's test, and in some respects a better one.

Take cupric sulphate, $36\frac{1}{2}$ grains ; potassic sodic tartrate, 178 grains ; caustic potash, 178 grains ; strong ammonia, 6 fl. oz. ; water, to 1 pint.

Dissolve the cupric sulphate by boiling it in one portion of the water, and the potassic sodic tartrate and the caustic potash in another ; then mix, and when cold add the ammonia, and finally the rest of the water. The advantage of this test is that the ammonia holds the cupric oxide in

solution. Ten c.cm. of the solution are equivalent to 0.005 gramme of glucose. (See Dr. Pavy, 'Lancet,' March 1884.)

(d) **TROMMER'S TEST.**—Add a few drops of a strong aqueous solution of sulphate of copper to (say) a drachm of urine in a test-tube. Then add caustic potash in such an excess as to redissolve the precipitate which it first forms. Then boil ; if sugar be present, a yellowish-red or 'telegraph-envelope'-coloured precipitate of cuprous oxide is formed. The presence of sugar can almost always be surmised, if after the copper sulphate and caustic potash are added the mixture is then of a deep transparent blue shade (Ord). If there be no sugar the mixture is generally a dull turquoise colour.

(e) **PICRIC ACID (Johnson's Test).**—The most convenient of all. To (say) a drachm of urine in a test-tube add an equal bulk of a saturated aqueous solution of picric acid as in the test for albumin ; then add the same amount of caustic potash. If albumin be present it will be redissolved by the alkali. Then boil ; the mixture, which was sherry-coloured, becomes mahogany-brown or almost black if sugar be present, the intensity of the colour varying according to the amount of sugar. Ordinarily the colour is not unlike that of a well-seasoned meerschau pipe.

Bile Acids.—**PETTENKOFER'S TEST.**—Mix thoroughly together, in a porcelain dish, an equal quantity of urine and a strong solution of cane sugar ; then gradually add, stirring with a glass rod the while, a similar quantity of strong sulphuric acid. A beautiful lake or purplish colour results if bile acids be present. This test can be conveniently modified by shaking the urine and syrup together in a test-tube so as to form a thick froth ; then gently pouring the acid down on the froth, which becomes purple-stained. In both methods the application of a little warmth will quicken the reaction. Any charring (black) of the sugar by the acid must not be mistaken for the purple colour of the successful experiment.

Bile Pigments.—To a few drops of urine in a porcelain dish add a drop of nitric acid, when a 'play of colours' results, the bilirubin being converted into biliverdin (Gmelin's test). The colours which are said to be observed are brown, yellow,

red, green, blue, violet, and dirty red. We must confess, however, that we have never seen this procession, and it is one of the most unsatisfactory tests we know of.

IODINE TEST.—To some urine (diluted if the colour be very dark) in a test-tube add a drop or two of tincture of iodine (one-third strength) by means of a pipette; this produces a green colour if bile pigment be present, bilirubin being converted by oxidisation into biliverdin. The watery tincture of iodine should be applied carefully so as to float on the surface of the urine.

Indican.—To form indigo-blue, mix with a given portion of urine in a specimen glass an equal quantity of pure hydrochloric acid; then apply one, or perhaps two (not more) drops of strong solution of calcic hypochlorite, and again agitate. When indican is present the resultant fluid is bluish, or even a dark bottle-green in colour. If chloroform be now briskly shaken up with it, it will absorb the newly formed indigo and become blue-stained.

A similar test applied to the urine of a patient taking iodides or bromides will produce a pink or claret-coloured staining of the chloroform.

To form indigo-red, boil a sample of urine and then add cautiously a few drops of nitric acid. This produces a rich red colour which will be taken up by chloroform on agitation.

Urea.—Mix equal parts of concentrated urine and pure nitric acid in a test-tube. After a time crystals of nitrate of urea are formed. The application of cold to the test-tube quickens the crystalline formation and deposit. If albumin be present in the urine, the nitrate of urea, instead of forming the usual needle-shaped crystals, is deposited in beautiful spheres, like snowballs, which gradually enlarge and, coalescing with neighbouring spheres, eventually fill the tube. (Ord, Rainey.)

The quantitative tests for urea are numerous.

(a) **HYPOBROMITE METHOD.**—This most convenient, and therefore the most popular, test depends on the decomposition of the urea by hypobromite of sodium into water, carbonic acid

and nitrogen, and the collection and estimation of the latter gas. Fifty-five c.c. of nitrogen correspond to 0.15 gramme of urea. Albumin, if present, must be precipitated and separated before using this test.

There are various forms of apparatus in use ; Russell and West's is best known, but is only convenient for laboratory work. A much more convenient ureometer, based on similar principles to the Russell and West, has been devised by Dr. Doremus of New York.

(b) **LIEBIG'S METHOD.**—This method is the most accurate, but the process is long and difficult. The basis on which it is founded is the chemical precipitation of urea by mercuric nitrate. For a description the reader is referred to the various text-books (Charles's 'Physiological Chemistry.')

Phosphates may be amorphous or crystalline.

The crystalline phosphates appear as the triple (ammonio-magnesian) and the phosphate of lime. The amorphous phosphates often produce a milky turbidity in freshly voided alkaline or feebly acid urines, which turbidity disappears or partially disappears on cooling, but is increased on boiling. Phosphates, therefore, are precipitated by heat, the precipitate being dissolved by the addition of acetic or other weak acid.

Oxalates.—Insoluble by heat or by acetic acid ; but soluble in hydrochloric acid.

Uric Acid.—Insoluble by heat, by acetic or by hydrochloric acids ; but readily dissolved by nitric acid and heat. Confirm by the following 'Murexide' test. Treat the crystals with nitric acid, and evaporate to dryness over a flame ; then apply ammonia vapour, when a beautiful pink colour results.

The following table gives easy test reactions of the principal crystalline deposits.

—	Apply heat	Apply heat and acetic acid	Apply heat and hydrochloric acid	Apply heat and nitric acid
Amorphous urates	disappear			
Phosphates . .	not affected	disappear		
Oxalates . .	—	not affected	disappear	
Uric acid . .	—	—	not affected	disappears

Cystine.—Insoluble in water and acetic acid ; but soluble in ammonia and hydrochloric acid.

Chlorides.—Add to a small quantity of urine in a test-tube an equal quantity of a solution of nitrate of silver (1 in 8). This will precipitate both ehlorides and phosphates. Then add a few drops of nitric acid, which redissolves the phosphates, leaving the chlorides as a dense, white preeipitate of chloride of silver.

We judge of their quantity by the bulk of the preeipitate ; if they are below $\frac{1}{10}$ or $\frac{1}{12}$ per cent. they only cause a milky eloudiness. The normal amount in urine is $\frac{1}{2}$ to 1 per cent.

Blood.—(1) **MICROSCOPE.**—Red blood-discs are recognised by their form, colour, and shape, all of which are preserved for some days in acid urine. They have no nuelei. They do not form rouleaux. In diluted urine they swell and lose their colour, and may thus be overlooked ; on the other hand, they may shrivel, or have crenated outlines, or present a broken, ‘bitten-out’ appearance of circumference. The absence of blood-discs is significant of hæmatinuria, provided there are signs of blood pigment.

(2) **GUAIAECUM TEST.**—Drop some of the urine suspected to contain blood on to a piece of clean, white filter-paper, then add tincture of guaiacum ; afterwards, when dry, apply a drop or two of ozonic ether ; if blood be present, a sapphire-blue colour results, especially at the edges of the stain. This test may also be applied by using the fluids in a test-tube. It is well to shake up the urine and guaiacum tincture first ; then pour the ozonic ether on to the froth, which momentarily becomes blue before it disappears ; and eventually the ether which floats on the top also becomes blue-stained. The presenee of saliva, pus, or iodine vitiates the test.

(3) **HELLER'S TEST.**—Add equal parts of urine and eaustie potash in a test-tube, and boil. If blood be present, the phosphates which are precipitated are stained a pinkish colour by the hæmoglobin.

(4) **SPECTROSCOPE.**—By proper manipulation the absorption bands of oxyhæmoglobin may be seen. Add a drop of saturated solution of hyposulphite of soda to the fluid containing

blood ; then apply a few drops of a strong solution of caustic soda. The hæmoglobin is thus converted into reduced alkaline hæmatin, the spectrum of which shows two absorption bands, one well marked between D and E. Note the presence of albumin in the urine if blood is in any quantity.

Pus.—The addition of caustic potash increases the viscosity or ‘ropiness’ of the urine, whilst it dissolves urates with which pus might be confused. The microscope shows a mass of granular, pale corpuseles, which clear up on the addition of dilute acetic acid.

In all cases note the quantity of albumin, as it may be excessive and out of all proportion to the amount of pus, and be thus symptomatic of other renal trouble. Renal casts in such cases should always be looked for.

BRIGHT'S DISEASE

Definition.—Non-suppurative inflammation of the kidneys, attended by albuminuria.

Under this heading several distinct conditions are recognised. *Clinically* they may be divided into two groups : ACUTE and CHRONIC. In the first group may be placed Acute Tubal Nephritis, and the sub-variety Scarlatinal Nephritis ; whilst the second will include Chronic Tubal Nephritis, or large pale kidney, and Chronic Interstitial Nephritis, of which latter the non-hereditary cystic kidney is, again, only a variety.

Pathologically the different varieties of Bright's diseases may be similarly classified into two groups, viz. : (1) those forms in which the pathological change is chiefly *intra*-tubular (acute tubal nephritis, large pale kidney, and fatty kidney) ; and (2) those in which the pathological change is chiefly *inter*-tubular (contracted granular kidney, and cystic kidney).

It must not, however, be supposed that in these affections the morbid changes are limited to the epithelium in one class, or to the interstitial tissues in another. It is probable that in any form of Bright's disease inflammatory changes, though

most marked, say, in the epithelium, are also observed in the interstitial tissue, and *vice versa*.

<i>Clinical</i>	TABLE OF BRIGHT'S DISEASES		<i>Pathological</i>
a. Acute	{ 1. Acute tubal or desquamative nephritis	{ Pathological changes principally <i>intra-tubular</i> , or <i>endothelial</i>	
	{ 1. Chronic tubal nephritis, or large pale kidney a. Fatty kidney		
b. Chronic	{ 2. Chronic interstitial nephritis, or contracted granular kidney a. Cystic kidney	{ Pathological changes principally <i>inter-tubular</i> , or <i>interstitial</i>	

ACUTE TUBAL NEPHRITIS

Definition.—An acute inflammatory disease of the kidneys, affecting the tubules principally, and attended by albuminuria.

Causation.—*Age.*—The disease appears to attack young people by preference. *Sex* has no influence. In adults it is usually secondary to *chills* and *exposures*, or a sequel to some *specific febrile disease*, such as scarlet fever, diphtheria, erysipelas, cholera, and ague. Or it may be induced by direct action of some *poisons* on the kidneys (turpentine, cantharides). It is not infrequently associated with *pregnancy*.

Pathology.—The kidney is slightly enlarged, and its capsule non-adherent. On section the organ is red; blood oozes from the cut surface; the cortex is relatively increased; dark vascular spots are easily seen by the naked eye (Malpighian tufts). Microscopically, a general hyperæmic condition of the organ is observed. The tubules are filled with (1) swollen and proliferated epithelium; (2) migrated blood-cells; or (3) fibrinous exudation from blood-vessels. Minute hemorrhages are frequent. Externally to the tubules we find proliferation of connective-tissue elements, some occasional red blood-cells, and also migratory leucocytes.

This disease may end in recovery, with absorption of all inflammatory products; or it may terminate fatally owing to uræmic poisoning; or it may become chronic, and pass on to

the condition known as chronic tubal nephritis (large pale kidney).

Symptoms.—The disease is ushered in suddenly, with some febrile disturbance, such as chill or rigor. Beyond this there are usually lumbar pains, vomiting, and headache. A pulse of high tension, with an accentuation and reduplication of the second sound over the base of the heart, draws our attention to the urine. This is scanty, often not more than eight or ten ounces daily, of high specific gravity (1030 or more), smoky colour, yielding abundance of albumin, and deposits on standing, free blood, casts (epithelial or blood), and amorphous urates. The albumin is much in excess of that which could be caused by the amount of blood in the urine.

The principal general symptom is dropsy, as shown by œdema of the eyelids, penis, and scrotum. This occurs early, and may be the first symptom of the disease. Then follow effusions into pleura, pericardium, and arachnoid; and, in the later stages, dry skin and pallor. Hæmorrhages (retinal, cerebral, petechial) occur in low debilitated subjects.

Prognosis.—Favourable, as a rule, if recognised early. Adults are said to recover in greater proportion than children. Our prognosis will be greatly influenced by the amount of urine excreted and the quantity of albumin it contains. Severe dropsy accompanied by gangrenous sloughing of skin are also symptoms of great danger. A recovery cannot be said to have taken place till the urine has regained its normal specific gravity, and all traces of albumin have gone. On the other hand, cases which are apparently hopeless recover even after some weeks of critical condition.

Complications.—Œdema of glottis; effusion into all serous membranes, especially pericardium and pleura; general dropsy; cerebral symptoms from uræmic poisoning, are the principal complications. The disease may pass into chronic desquamative nephritis. Erysipelas is not infrequent.

Treatment.—The patient must be kept absolutely at rest in bed and between blankets till all traces of albumin in the urine have vanished. The diet should consist of milk, eggs, and farinaceous foods only. Alcohol in all forms must be

forbidden. In mild cases this diet and regimen will often be sufficient without medicinal aid. Should medicines be required, give such remedies as promote elimination by skin, bowels, and lungs, and so relieve the kidneys. Leeches may be applied to the loins in robust patients, followed by hot fomentations or cupping, hot packs, and diluent warm drinks. *Liquor ammoniæ acetatis* is of great value in the early stages.

To act on the bowels we may order the saline purgatives (sulphate of magnesia or of potash), or compound jalap powder. We may in urgent cases resort to colocynth and gamboge ; and at the same time promote diaphoresis by the hot vapour-bath and the internal administration of *jaborandi*.¹

Diuretics are not without danger in the early acute stages, but may be given with advantage if the disease tends to linger or run into chronicity. At this time copious libations of warm water will frequently ' wash out ' any remnant of disease yet lingering in the tubules.

Avoid mercurials, and also turpentine and cantharides.

During convalescence, iron with squill and *digitalis* are of great service.

CHRONIC TUBAL NEPHRITIS (CHRONIC DESQUAMATIVE NEPHRITIS ; LARGE PALE KIDNEY)

Definition.—A chronic, inflammatory disease of the kidneys, affecting principally the epithelium of the tubules, and attended by albuminuria.

Causation.—This form is, in most cases, a sequel of acute tubal nephritis ; but it may originate in some general condition producing disordered health, and in such cases appears to be a chronic disease from the onset. The chief predisposing causes are : *Age*.—Most cases are seen in middle-aged adults. *Occupations* involving exposure to changes of temperature. *Diet and Habits*.—There is often a history of alcoholic excess, or of unwholesome food and surroundings. *Poisons* of various characters which are used in arts and manufactures, such as phosphorus, mercury, are

¹ *R.* Ext : *Jaborandi* gr. iij. ; Ext : *Gentianæ* gr. ij. Miscæ ; ft. pil.

thought to produce the disease. But the evidence is not conclusive.

Pathology.—The changes are chiefly found in the kidney tubules and their epithelium.

The kidney is large, showing a tendency to lobulation ; the capsule peels easily, leaving a smooth, pale surface, often crossed by stellate veins. On section the cortex is seen to be increased, and pale in colour, whilst the medullary cones are injected and red ; occasionally small hæmorrhagic points are found in the cortex and the medullary part also.

On examination by the microscope, the tubules are found distended, and also less tortuous than in health ; their walls are thin, and the epithelium lining them is large, swollen, and undergoing granular degeneration ; the original outline of the epithelial cell is often lost, the nucleus is absent or difficult to find. Here and there the epithelium may be shed in blocks, which fill up the lumen of the tubule ; or it may be necrosed in patches and washed away entirely, leaving only a bare basement membrane. The Malpighian tufts are also compressed by their own proliferated cells, and the capsules themselves are thickened. There is in addition some evidence of inflammatory process in the intertubular tissue.

FATTY KIDNEY.—In later stages, when fatty degeneration has occurred, the organ retains the principal features described above ; but in addition it is greasy to the touch, whilst the epithelium becomes filled with fat-granules, or indeed replaced entirely by oil-globules.

Some authors describe yet another condition of pale kidney in which the intertubular tissues become the seat of more marked inflammatory changes. These changes produce contraction of the connective tissue, by which the organ becomes slightly granular, the condition apparently resembling that of not advanced interstitial nephritis.

Symptoms.—There is general debility with marked facies, which is almost an index to the condition of the kidney. The face is pale and pasty, with puffiness of eyelids, producing a heavy expression. The skin is dry and very pallid, there is a general tendency to dropsical effusions into serous cavities

and into the subcutaneous areolar tissues, especially of eyelids, scrotum, and ankles. Prior to these marked signs the patient notices that he has dyspnoea supervening on little or no exertion, and a polyuria. The urine on examination is clear, pale-coloured, of specific gravity, as a rule, below normal; it contains much albumin, and deposits renal epithelium which shows degenerative signs, leucocytes, and casts which are mostly granular or fatty.

The symptoms may be complicated by occasional attacks of acute tubal nephritis; the urine will then present the mixed characters of both conditions. As the disease progresses, hypertrophy of the heart and some of the vascular changes of contracted kidney supervene. The disease is a progressive one, but is often characterised by periods of apparent amelioration.

After a time general dropsy may supervene, with fatal œdema of the glottis. Uræmia, due to retention in the system of substances which should be eliminated by the kidneys, occurs in the final stages.

Prognosis.—Favourable if recognised and treated in the early stages; but when the disease is once fairly established it is invariably fatal. Yet life may be prolonged for some years by careful attention to diet and surroundings.

Diagnosis.—1. FROM ACUTE TUBAL NEPHRITIS.—By the duration of the disease, the aspect of the patient, and by the chemical and microscopical evidence afforded by the urine (see pp. 517, 523).

2. FROM LARDACEOUS DISEASE.—By the absence of any history of syphilis or suppuration, and by the physical signs of lardaceous disease in the liver, spleen, or elsewhere.

3. FROM CONTRACTED GRANULAR KIDNEY.—The patients with this condition are generally older; there is often a history of gout; and the cardio-vascular changes are more pronounced. The urine is also of low specific gravity, and contains often a trace of albumin only (see p. 542).

4. THE ALBUMINURIA OF HEART DISEASE is distinguished by the chronic history of the case—an illness much longer than that of large pale kidney; by the evidence of

valvular or other cardiac lesion ; and by the presence of casts and epithelial deposits in the urine to a much less extent than in chronic tubular nephritis.

Complications.—**SEROUS EFFUSIONS** into the pleura and pericardium are common in all forms of chronic renal disease. **BRONCHITIS** is also a frequent complication, especially in the advanced stage of the disease. **HÆMORRHAGES** may occur from the nose or from the retinal vessels, or within the brain, but less frequently than in contracted granular disease of the kidneys.

As regards the skin, it is liable to inflammatory affections and bedsores from pressure ; sloughs due to anasarca may also take place. **URÆMIC CONVULSIONS** and **COMA** are the usual complications which precede death.

Treatment.—The following general rules should be observed.

The diet should consist of milk and farinaceous foods principally. Little or no animal food should be allowed, except it be occasional fish or poultry. No alcohol whatever is to be permitted, unless temporarily when urgently required. Warm flannel clothing is essential. The skin and bowels must be encouraged to act freely. The patient should reside in a warm equable climate if possible.

Tonics which improve the condition of the blood, such as iron salts, quinine, arsenic, may be prescribed with advantage.¹

Diuretics (squill, digitalis, acetate of potash, scoparium), are of good service if given for a length of time. No mercury should be prescribed. It may be necessary in advanced cases to puncture œdematous legs, or to use Southey's trocars. This treatment, however, tends to produce sloughs unless guarded by strict antisepsis. It is best avoided if possible.

Poultices may be applied to the loins with digitalis leaves added, if there are signs of acute exacerbation of the disease.

Promote the action of the skin by liquor ammoniæ acetatis and hot-air baths. Hydragogue cathartics (pulv. jalapæ comp.) are useful from time to time.

¹ ℞. Tinet: Ferri Perchlor: ℥xv.; Tinet: Digitalis ℥x.; Tinet: Scillæ ℥xx.; Aquam ad ʒj. Misce.

CHRONIC INTERSTITIAL NEPHRITIS (CONTRACTED GRANULAR KIDNEY ; GOUTY KIDNEY)

Definition.—A chronic inflammatory disease of the kidneys, affecting principally, and causing an increase of, their intertubular connective tissue, followed by atrophy of these organs.

Causation.—*Age.*—The disease occurs most frequently between forty and sixty, yet many cases are recorded in early life. *Sex.*—A great preponderance of cases is found in males. *Heredity* is often a well-marked feature. Numerous cases of transmission from father to son are recorded, although both were abstemious in eating and drinking. *Alcohol* probably causes a slight percentage of cases ; but its influence is by no means settled. *Gout* is a most potent factor. A chronic gouty inflammation of a joint portends contracting kidney.¹ *Rheumatism.* *Chronic lead poisoning* is conducive to the disease, inasmuch as it is associated with gout, inflammation of joints and the surrounding fibrous tissues.

Pathology.—The kidneys are small, occasionally no larger than walnuts. The atrophy may be much more pronounced in one kidney than in the other. The capsules are adherent and peel off with difficulty, leaving coarse, granular and tawny-coloured surfaces on which, it may be, small cysts are scattered. On section the organ is pale, and of tough consistence ; the cortex is atrophied and often consists of a shallow rim only, surrounding the pyramids ; it may or may not contain cysts. *Microscopically* the kidney structure is seen to be infiltrated, especially in the cortical portion, by a fibro-nucleated growth, a hyperplasia of the connective tissue between the tubules. The earlier and more rapid the disease, the greater the preponderance of nuclei ; whilst the chronicity of the change is marked by an exuberance of fibroid tissue,

In seventy-three post-mortems observed in gouty subjects the presence of uratic deposit in any joint was invariably accompanied by cirrhosis of kidneys.—S. T.

which, by its shrinkage, compresses the tubules and thus produces irregularities in their course, shape, and lumen.

The Malpighian tufts are reduced in size ; the capillary constituents are shrunken and matted together ; and the surrounding capsule is always thickened, and sometimes laminated, though often of homogeneous structure.

According to Gull and Sutton, there exists 'a fine hyaline fibroid substance between the tubules, making them appear further apart than normal. A similar change occurs in the arterioles throughout the body, especially situated immediately outside the muscular layer.'¹

The cystic kidney is, unless congenital, probably an extremely chronic form of the contracted granular kidney. The cysts result from retention of the contents of tubes or Malpighian bodies, caused by obstruction due to the adventitious fibrous tissue.

Symptoms.—The disease is essentially a chronic one, and comes on insidiously. There is a typical facies ; the features are often sharp ; the expression is anxious, yet the complexion is fresh, if not florid. Probably the first symptom complained of is dyspnœa on the slightest exertion. The patient's attention is then drawn to the urine, owing to his having to rise once or twice in the night. He frequently has dyspepsia ; the bowels tend to constipation.

Neuroses then become common, viz. : headache and vertigo, irritability of temper, and a failing memory ; whilst the ophthalmoscope reveals hyperæmia of the optic discs in consequence of serous exudation. It may be followed by retinal hæmorrhages, or atrophy of the discs.

When the disease is fairly established, symptoms pointing to change in the cardio-vascular system are the most pronounced. 'Hypertrophy of the left ventricle is an early sign. This hypertrophy is not dependent on the kidney change, but is due to morbid alterations in the vessels, not only of the kidneys, but of the body generally. The disease, therefore, is not necessarily originated in the kidneys.' (Gull and Sutton, *op. cit.*) The pulse is one of high tension ; there is a tendency

¹ *Trans. Med. Chir. Soc. Lond.* vol. lv.

to palpitation on exertion, and to hæmorrhages from mucous and other surfaces. Epistaxis is most common, and, on the whole, perhaps is beneficial rather than otherwise. Bleedings, however, may occur from the stomach or bowels; also on the surface of the brain, or into its ventricles or substance, thus causing apoplexy. Retinal hæmorrhage with total blindness is not uncommon.

The urine is copious, amounting often to four or five pints a day; it is almost colourless; its specific gravity is low (1010, or less); it contains a little albumin, but often none, and deposits hyaline and granular casts, epithelial cells, and, occasionally, free blood. Urea is scanty in an individual specimen.

The skin is often dry and scaly, and there is a diminution of subcutaneous fat; anasarca is not common, at least in the early stages of the disease. Attacks of bronchitis are frequent, and when the disease has advanced there is a liability to pneumonia.

Complications.—Besides the cardio-vascular conditions, which are really part of the disease, other complications involving the respiratory and nervous systems are common. Dyspnoea is often urgent ('asthmatic' attacks), and occasionally aggravated by œdema of the glottis. Albuminuric retinitis with total blindness, in some instances one of the earliest symptoms, is found in the later stages. Delirium, uræmic convulsions, and coma are often present towards the end, which may be preceded by ascites and other dropsical effusions.

Treatment.—Our aim should be directed chiefly towards reducing the arterial tension, and so to prevent dangerous hæmorrhages. The diet should be plain and nutritious, and consist principally of milk, eggs, and farinaceous foods. Animal food must be allowed sparingly only. Alcohol is strictly forbidden.

Nitro-glycerine, in doses of $\frac{1}{100}$ gr. twice a day, is of great service in relieving tension in the smaller vessels. We may also give saline watery purgatives, such as the sulphates of magnesia, potash, and soda, in combination with iron or quinine. For uræmia, opium in small doses has been proved

to be of good service, in spite of theoretical objections. The subcutaneous injection of pilocarpin (gr. $\frac{1}{16}$) should be administered if convulsions supervene. Vomiting is best relieved by dilute hydrocyanic acid, with effervescing draughts.

Warmth is an important item. The patient should wear flannel constantly, and, if possible, should winter abroad, or avoid the dangerous winds which prevail in spring. He should also obey the ordinary laws of health as regards occupation, exercise, &c.

TABLE OF URINE IN BRIGHT'S DISEASES

—	Acute Tubal Nephritis	Chronic Tubal Nephritis	Chronic Interstitial Nephritis
Quantity	Scanty . . .	Increased . . .	Much increased
Colour .	Smoky . . .	Pale yellow . .	Almost colourless
Sp. gr. .	Increased (1030)	Diminished (1015)	Much diminished (1010)
Albumin	Loaded . . .	Much . . .	Little or none
Deposit .	Free blood . .	Occasionally free blood	Occasionally free blood
	Renal epithelium	Degenerated epithelium	Epithelium
	Casts { epithelial blood granular	Casts { granular fatty hyaline	Casts { hyaline granular

It seldom occurs in chronic Bright's disease that one single type of kidney lesion is found. The mixed kidney, showing either acute tubal combined with chronic tubal changes, or the contracted kidney, with co-existing, chronic, epithelial lesions, is the most frequent condition.

PYELITIS

Definition.—Inflammation of the pelvis and infundibula of the kidney.

Causation.—Generally secondary to some local irritation, such as impacted calculus in the kidney or ureter, chronic cystitis, prostatic disease, or urethral stricture. It may supervene on the administration of turpentine, cantharides, or other irritant drug. Or it may result from tubercular disease of the

urinary tract. A gouty form of pyelitis, due to the constant irritation of very acid urine, is also described. Occasionally it is a complication of some of the specific fevers, especially pyæmia.

Pathology.—There is primarily congestion of the blood-vessels supplying the part; the pelvic mucous membrane becomes swollen with proliferated epithelium. The pelvis then dilates, its walls become thickened, shed their epithelium, and secrete a muco-pus. As the disease advances, the pelvis becomes an abscess cavity, with pouches or diverticula corresponding to the original infundibula. Its mucous membrane is grey-coloured and sloughs in patches.

Should complete obstruction occur, a globular, tense tumour forms, containing pus (pyonephrosis), which may be evacuated by the urinary tract or by fistulous openings into the peritoneum, the colon, or through the loin.

Symptoms.—Such as would accompany an abscess in the kidney, viz.: a high temperature, hectic fever with rigors, sweats and vomiting, accompanied by a dull and continuous pain radiating from the loins to the umbilicus, groins, thighs, and penis. There is frequent desire to micturate, which temporarily relieves the pain. The penis and testicles are usually retracted. The urine, unless complete obstruction occurs, contains blood, conical or pear-shaped epithelium from the pelvis, mixed with pus and shreds of mucous membrane. It may also contain phosphates from decomposition; but usually the reaction is acid and slightly albuminous. If tubercle be suspected as primary cause, the urine should be searched for bacilli.

Diagnosis.—FROM PERINEPHRIC ABSCESS.—This condition would be attended by severe pain and more marked tenderness, which is comparatively superficial. Nor does the urine contain pus necessarily, although there is frequently a certain amount of albuminuria.

FROM HYDRONEPHROSIS.—This retention tumour occurs usually without severe pain, except on exertion, and without marked pyrexia. It may also disappear from time to time as the obstruction is removed or alters its position. The urine is

free from pus, unless the obstruction is chronic and causes a secondary pyelitis.

Prognosis will depend on the cause and the duration of the disease. Tubercular pyelitis is ultimately fatal. A similar remark applies to those cases in which a fistulous opening allows the suppuration to become chronic and thus cause lardaceous disease. Cases, however, recover after removal of the diseased kidney, and in the mild forms which occur secondarily to fevers.

Treatment.—In the acute stage : local applications to the loins of hot poultices or fomentations ; leeches or wet cupping. Medicinally, saline febrifuges and purgatives are indicated. Give opium to relieve the pain. The removal of the cause of primary irritation, whether it be in urethra, bladder, or kidney, may demand surgical interference. Free drainage of accumulated pus is essential, as the abscess, if left alone, is almost invariably fatal. The after-treatment demands tonics, a nutritious diet, and a bracing climate.

LARDACEOUS DISEASE OF THE KIDNEY

Definition.—A degeneration secondary to prolonged suppuration, syphilis, or bone disease, involving the kidneys in common with the liver, spleen, and other organs.

Causation.—*Age.*—Cases more frequently occur in young and early adult life. *Wasting and suppurative diseases*, such as phthisis, caries of bone, and more especially tertiary syphilis.

Pathology.—The kidney is enlarged, of pale, waxy appearance, and firm to the touch. The capsule easily peels off. On section the cortex is enlarged, and presents shiny, glistening points, resembling the cut surface of fat bacon or suet. The degenerative change commences in the muscular coats of the smaller blood-vessels. The capillary Malpighian tufts are the first to be involved ; the disease then spreads to the larger arteries and veins. The walls of the urinary tubules and the intertubular vessels become affected later. The tubules themselves contain, and are distended by, swollen

epithelium and epithelial débris, and perhaps also by fatty matter in the secondary and later stages. Large hyaline casts are almost invariably found plugging the larger tubules.

The iodine reaction is the typical test. On applying a weak solution of iodine to a freshly-cut surface of the kidney, it attacks the Malpighian tufts, and also the larger vessels, so that they can be easily seen as dark mahogany-coloured points or lines. Methyl-violet also presents a contrast stain by turning the amyloid tissues a pinkish-red and the rest of the organ a blue tint.

Symptoms.—There is a previous history of syphilis, chronic abscess, or caries of bone. There is marked pallor, together with harsh and ‘muddy’ skin. The urine, voided in large quantities, is of low specific gravity (1008), pale in colour, contains a fair amount of albumin, and deposits large, brittle, hyaline casts. In the later stages only, there may be œdema of legs, and also ascites. Death usually occurs under twelve months from the commencement of the disease, and is directly due to exhaustion, or to diarrhœa as a result of lardaceous changes in the mesenteric vessels.

Uræmic symptoms are not common ; nor are there cardiovascular or retinal changes as in chronic Bright’s disease. The spleen and liver are almost invariably and contemporarily involved.

Diagnosis.—This is usually determined by the history of suppuration, the presence of signs of lardaceous disease in the spleen and liver, and by polyuria with a larger amount of albumin than in contracted granular kidney, and less than in chronic tubular nephritis.

Treatment.—Entirely secondary to the disease of which it is a complication. All we can hope for is to prolong life, by the administration of cod-liver oil, iron and other tonics, and to relieve complications which may arise.

Treat diarrhœa and dropsical accumulations on broad general grounds. A nutritious diet is also indicated.

HYDRONEPHROSIS

Definition.—A non-inflammatory distension of the ureter and of the pelvis of the kidney by obstructed urine.

Causation.—(1) The obstruction may be exerted by pressure from *without*—*e.g.* new growth in relation with the bowels, lymph glands, bone, muscle, uterus, or other structure in the vicinity of the kidney and ureter. (2) Obstruction of the ureter may occur from *within*, such as by new growth, calculus, and thickened walls of the bladder. A calculus may be lodged in the ureter itself ; occasionally a congenital valve or fold of mucous membrane, or a twist exists in the ureter. Hæmorrhage into the dilated pelvis or into the ureter is a comparatively rare cause. Hydronephrosis is more frequent in women than in men, whatever the cause may be.

Pathology.—The kidney may be little altered in shape or structure in small accumulations. On the other hand, the sac may assume huge proportions, distending the pelvis, infundibula, and calices to such an extent that the kidney is scarcely recognisable beyond its being a large irregularly sacculated structure, with traces only of the cortical portion remaining as a cyst wall. Suppuration of the sac may occur, or it may ultimately shrivel and its contents become absorbed. Hydronephrosis is occasionally found in both sides, and is then generally congenital ; or it is due to some disease in the bladder or urethra by which both ureters are obstructed. Similarly the ureter may be distended till it reaches the diameter of a large sausage, or larger. Obviously, if the obstruction is near to the bladder the whole of the ureter will be equably dilated.

Symptoms.—No severe symptoms. There is no pain or discomfort except that arising from the tumour formed by the accumulation. This tumour, situated over the region of the kidney and ureter, is of rounded or oval shape, smooth, and generally tense to the touch. Fluctuation may generally be easily made out. It is dull on percussion, except anteriorly, where it is overlapped by the colon. It is liable to sudden

disappearance, attended by an increased flow of urine, owing to the partial removal of the obstruction or its sudden alteration in position. Unless the obstruction, however, be entirely removed, the fluid again collects and the tumour reappears. The signs of pressure on neighbouring organs are not very definite unless the cyst assumes large proportions. The urine itself is little altered. It may contain a tinge of albumin; but pus is absent as a rule. The fluid of the cyst consists of urine with only traces of its urea and salts. In other words, the kidney structure has been damaged by the distension, and almost ceases to excrete solid matters.

Diagnosis.—FROM PYONEPHROSIS.—The physical signs are much the same in both diseases. In hydronephrosis, however, we should be guided by the absence of marked fever and other constitutional symptoms, and by the freedom of the urine from much constant pus.

FROM PERINEPHRIC ABSCESS.—This condition is accompanied by much local pain and tenderness, by rigors and fever. The disease also is of longer duration in reaching the stage of physical signs than hydronephrosis.

FROM OVARIAN TUMOUR.—This tumour grows up from below, manifestly in connection with a pelvic organ. Internal examination also will show that the uterus is displaced.

AN HYDATID CYST is extremely tense; it pushes every structure before it, even bulging the ribs. Aspiration and examination of the fluid will reveal the presence of hooklets.

A fluctuating tumour, situated in relation with the kidney or the ureter, which suddenly subsides and subsequently reforms, can only be hydronephrosis.

Prognosis.—Favourable in the majority of cases. The dangers which may arise are, pressure on some vital organ, or rupture of the cyst and discharge of its contents into the peritoneum. If both sides are affected, the gravity of the case is obviously increased. Occasionally, after aspiration of the cyst, a fistulous opening may be kept up by the discharging fluid, and so lead to death from exhaustion unless the kidney be excised.

Treatment.—Surgical entirely. Puncture the cyst with

due precaution to anatomical relations. Kneading, with a view to dislodging the obstruction, has also been advocated.

MALIGNANT DISEASE OF THE KIDNEY

Any form of malignant new growth may primarily attack the kidney. Sarcoma and carcinoma are the most common. These diseases, however, are rarer than in the liver or alimentary canal. The kidney is not infrequently involved by secondary deposits from suprarenal bodies, bowel, or other neighbouring organ.

Causation.—*Age.*—Encephaloid cancer occurs most frequently in adults, whilst the sarcomata are oftener met with in young patients. *Heredity* may have some predisposing influence. *Sex.*—Men are said to suffer more frequently than women. *Injuries* also act as predisposing causes ; under this category we must also place the irritating effects of renal calculi. The right kidney is oftener involved than the left.

Symptoms.—Loss of flesh is an early symptom, and precedes any other ; then follows pain in the loins, often of a severe, lancinating character, and extending to the groin and thigh. The urine from time to time contains blood freely mixed, and frequently deposits epithelioid cells of cancer or sarcoma. The cachexia common to malignant disease, wherever it occurs, shows itself sooner or later. The diagnosis is further completed by percussion and palpation revealing a generally hard tumour situated behind the colon, which gradually increases in bulk so that it may assume huge proportions, and thus give rise to a distension of abdominal veins, œdema of the lower limbs, and even paraplegia. The urine does not necessarily contain albumin, unless there is hæmaturia. This latter, as stated above, is an intermittent symptom, the urine often being quite normal between the attacks. Secondary infiltration of lymph glands should also be looked for.

Diagnosis.—The existence of a renal tumour occurring in an elderly subject, and accompanied by intermittent hæmaturia, and wasting, would render the diagnosis of malignant disease

of the kidney almost certain. But without hæmaturia the diagnosis is more difficult, as the tumour may be confounded with similar or other conditions in connection with *liver* or *spleen* or *bowel* or *ovary*. A tumour originating in either liver or spleen is separated from the kidney by an area of resonance, and it should move with the ascent and descent of the diaphragm, unless considerable adhesions to surrounding parts have occurred. In malignant disease of the colon the ordinary resonance of the bowel over the kidney would be lost, and in addition there would be evidence afforded by the evacuations. Ovarian tumours grow from the pelvis, and are associated with displacement of the uterus. Scybala may at times be felt on palpation ; but this condition is not preceded by emaciation, and a large enema would at once clear up any doubt.

As regards the diagnosis between malignant disease and other tumours of the kidney itself, these, mostly containing fluid, yield fluctuation on palpation ; they are not so irregular in their outlines as cancerous growths, and, as a rule, the distress they afford is one of centrifugal pressure and discomfort rather than the severe pain of malignant disease. An exploring needle would materially assist in diagnosis.

The examination of the urine for the detection of cancer cells is apt to be misleading, and is indeed unreliable, owing to the varying types of epithelium which occur in the different parts of the urinary tract.

Treatment.—Palliative only. Give opiates and morphia to relieve the pain. Treat hæmaturia on general grounds. Attend to the patient's health. His diet should be nutritious and generous. If the question of nephrectomy be entertained, it should be attempted early. It is useless in advanced stages, or if there be secondary infiltration of other parts.

TUBERCULOSIS OF THE KIDNEY

Causation.—*Age.*—The acute form is more common in early life. The chronic variety may occur at all ages. *Sex.*—Men suffer more frequently than women. The other pre-

disposing causes are much the same as produce tubercular invasion of other organs. By some authorities it is supposed that the presence of renal calculus in a strumous subject may act as an exciting cause to tubercular deposits in the kidneys.

Pathology.—The disease may be a primary or a secondary affection.

In *Primary* tuberculosis of the kidney the lesion first appears in one or the other gland, or at least is quite as far advanced here as in any other organ.

Aggregations of tuberculous growths which are formed by the coalescence of neighbouring tubercles are found in both the cortical and the pyramidal zones of the kidney. These, like similar growths elsewhere, undergo retrogressive caseation and softening, and so produce a series of excavations which may remain discrete, or which may be connected by ulcerous intercommunicating channels.

Each cavity is lined by a pyogenetic membrane, studded with tubercles, and filled with cheesy pus and detritus, with some lime-salts. In advanced cases, the excavation goes on to such a degree that the organ is practically destroyed, and is represented by a large abscess, with somewhat thick walls, in which only here and there a remnant of the gland tissue remains. The disease may be unilateral; but generally both kidneys are involved.

Usually, also, a similar dissemination of tubercular deposits occurs in the pelvis and infundibula of the kidney, which become dilated, and also in the ureter, which is thickened and almost obliterated. The resultant condition therefore resembles that of pyonephrosis. Tuberculous ulceration of the bladder and of the vesiculæ seminales is also frequent, probably from infection brought down from the kidney.

In the *Secondary* form of the disease the kidney may be the seat of miliary tubercular deposit as part of a general acute tuberculosis.

Symptoms.—In their main features bear a resemblance to those of pyelitis and new growth. The patient complains of

a dull aching pain in the loin, accompanied by fever which is generally of the hectic type. He has frequent micturition, the urine containing pus, shreds of epithelium and of kidney tissue, and the characteristic tubercle bacilli. Tube casts may be present, but not necessarily so, as the renal tubules and epithelium are not primarily involved. The urine is generally of acid reaction ; but it may be alkaline and contain an excess of pus if tuberculous cystitis be present.

Beyond these symptoms the patient loses flesh and strength. The lungs and other organs generally also afford evidence of tuberculous disease, in a more or less advanced state.

Diagnosis.—As the signs are practically the same as in pyonephrosis, we are only led to suspect tubercular disease of the kidney by finding a similar condition in the lung, testes, or elsewhere. A microscopical examination of the urinary deposit and the discovery of the tubercle bacillus would render the diagnosis certain.

Prognosis.—Always unfavourable. The length of life depends on the previous duration of the disease, and the integrity of the fellow-kidney.

Treatment.—The local treatment is the same as in pyelitis. The question of nephrectomy may have to be considered. It is hopeless, however, in advanced cases in which amyloid degeneration is a complication ; and must not be entertained even, unless the disease is limited to one kidney.

The general treatment consists in maintaining the appetite and strength of the patient by tonics and cod-liver oil.

MOVABLE KIDNEY (FLOATING KIDNEY)

Definition.—A pathological condition under which the kidney is dislocated from its normal situation and manifests more or less mobility.

Causation.—*Age.*—The greatest number of cases have been observed between the ages of thirty and forty. The right kidney is most frequently found displaced. *Sex.*—Nearly eighty-seven per cent. of cases are females ; the

majority of these being women of the working classes, who have borne large families.

Other causes may be named, such as tumours of the supra-renal bodies, or of the pancreas, disease of lumbar vertebræ and psoas-abscess, direct injury to the kidney, absorption of its fatty capsule, tumours and displacements of the uterus, and a general lax condition of the walls and tissues of the abdominal cavity. There is often a previous history of emaciation following on obesity.

It is occasionally congenital, due to anatomical abnormality in which a mesonephron is developed.

Symptoms.—There are not necessarily any marked symptoms or inconvenience. The patient has generally, however, a sense of pain and a dragging discomfort in the loin, accompanied by some nervous disturbance, such as hypochondriasis or a local neuralgia. There is often derangement of digestion and disorder of the gastro-intestinal canal. There may be suppression of urine, or even hydronephrosis, owing to twisting of renal vessels and duct; and the expulsive efforts attending parturition, defæcation, or micturition, often give rise to pain, nausea, and fainting. Occasionally œdema of the lower limbs occurs from pressure on the inferior cava.

Palpation, as a rule, easily reveals the displaced organ according to the extent of its mobility. In some cases it may be so manipulated as to be pushed over the lumbar vertebræ. If the trunk be raised, the kidney may be felt extending into the pelvic fossa. The patient should be examined bi-manually and in different postures; the best position, however, is the 'knee-elbow,' with the trunk slightly raised. The kidney will then fall towards the abdominal wall, leaving a hollow depression in the loin. The organ is known by its shape and smooth surface; it easily slips from under the fingers; manipulation is generally painful.

Diagnosis.—This entirely rests on the movable character of the tumour, its rounded, smooth surface, and the faint sickening sensation which it produces on manipulation. Further, the tumour rarely increases in size, and when restored to its normal situation it is overlapped by a resonant bowel.

Prognosis.—Good. It is rarely fatal if left alone. The prominent symptoms being of a neurotic kind often disappear as soon as the innocent character of the tumour is established in the patient's mind.

Treatment.—To restore the kidney to its proper site and retain it there by (1) a firm, broad, flannel binder round the whole abdomen ; (2) a concave metal shield fixed over the organ ; (3) corsets covering the whole of the belly down to the groins. Strengthen the muscles of the abdominal wall by massage, electricity (induced current), sea-bathing. Prevent constipation, vomiting, or other violent expulsive efforts.

Surgical interference has hitherto not been markedly successful ; but the operation by which the kidney is attached to the posterior abdominal wall (nephrorrhaphy) may have to be considered in cases in which there is much distress. (Landau, New Sydenham Society, 1884.)

RENAL CALCULUS

Definition.—A concretion of uric acid, oxalate of lime, phosphates, or other crystalline matters, which lodges or is formed in the pelvis or infundibula of the kidney.

Pathology.—The calculi vary much in composition, shape, size, and situation.

Uric acid concretions are the most frequent. The stone may be only just too large to pass down the ureter, or it may be the size of a pigeon's egg. Its form is generally oval or rounded, but often branched, and closely fitting to the divisions of the infundibula. In some cases numerous calculi exist, which are then faceted by mutual attrition. They are always deposited in acid urine.

Oxalate of lime.—These calculi are generally small, tuberculated, of extreme hardness, and black on the surface from decomposed blood. They are deposited in acid or neutral urine.

Phosphatic concretions in the kidney are not frequent, except as secondary incrustations on previously-formed calculi which by their obstruction have caused decomposition of

urine. They are generally smooth and of dusty or chalky appearance, and often attain a large size. They usually are composed of calcic and ammonio-magnesian phosphates mixed, and are deposited in alkaline urine.

Cystine calculus is rare ; it is light, and of a peculiar, burnished, waxy, or sooty exterior. It has a radiating and glistening appearance on section, and is deposited in acid urine.

Other calculi which may form are, the carbonate of lime, the xanthine, and the urate of soda. Dr. Ord has discovered a calculus which was composed of indigo.

The origin of all calculi is still obscure. The nucleus may be formed of blood, parasitic ova, epithelium, mucus, or pus. Having thus a starting-point, a preponderance of any of the crystalline bodies so frequently found in urines would tend by concretions and laminations to the formation of a calculus. It is not common for a given calculus to be composed entirely of one urinary product. A mixed calculus with alternating layers is far more usual.

The secondary results of a calculus are, pyelonephritis, hydronephrosis, pyonephrosis.

Disruption of calculi may occur (according to Ord) owing to some alteration, mechanical or chemical, in the medium in which they are found.

Symptoms.—The symptoms of renal calculus vary according as it remains stationary or passes down the ureter. We may thus have symptoms which are significant of (*a*) renal irritation ; others which are due to (*b*) obstruction and spasm of the ureter ; and, finally, there may arise symptoms which point to (*c*) disorganisation and destruction of the affected kidney.

(*a*) If the calculus rest in the kidney or its pelvis, symptoms are not always prominent. But ordinarily pain is a prominent symptom, although a renal calculus may exist for years and give rise to no suffering. It consists of a dull, heavy, and aching pain in the loin, aggravated by jars and joltings, and occasionally inducing vomiting. The urine may contain small concretions, or crystals of the calculus, but always free blood at some time or other. The hæmorrhage may not be

sufficient to colour the urine, but it invariably is present in one or other specimen of urine if searched for microscopically. Occasionally also the urine contains pus or albumin.

(b) If the calculus be small and it become dislodged from the pelvis and travels into the ureter, there would occur all the signs of renal colic. We ought then to have every sample of urine searched for a calculus. The stone in its migration always causes well-marked symptoms, viz. pains in the lumbar region, which are constant for some hours, or paroxysmal and more accentuated in the side where the calculus is lodged. They also radiate to the belly, the loins, the buttocks, and thighs. There is often also urethral pain, with retraction of the penis and testicle, and a frequent desire to micturate. Nausea, vomiting, and rigors are common, as in biliary calculi. No fever exists, as a rule, unless the attack be a long one; but, on the other hand, a feeble pulse and a pallid skin point rather to symptoms of shock.

The urine contains free blood, and deposits crystals according to the composition and structure of the calculus. All these symptoms abate as the calculus passes into the bladder or escapes by the urethra.

(c) The subsequent results of a calculus which is impacted in the pelvis or in the infundibula of the kidney are, pyelitis, pyonephrosis, and possibly perinephric abscess. The general symptoms which would indicate these conditions are, constant lumbar pain, the presence in the urine of pus and blood, and possibly phosphatic crystals, with hectic fever, and emaciation.

Diagnosis.—The presence of lumbar pains, hæmaturia, and an abundance of one or other of the typical crystals in the urine, with absence of any definite renal tumour, are strongly suggestive of renal calculus, especially if the urine remain acid. These conditions may be simulated by cancer of the kidney; but in this latter disease, which occurs more frequently in advanced life, the hæmorrhage is more profuse. A history of gout also would point to calculus as the cause of the symptoms. Frequently a positive diagnosis cannot be determined until an exploratory incision be made in the loin and a needle used as a sound.

Treatment.—Correct the diathesis. Benzoate of ammonium is an invaluable remedy when the urine is alkaline from irritation of any retained calculus.¹ Excess of uric acid should be met by large doses of alkalis (carbonate of soda or potash), with a view to solvent effects of an alkaline urine. The dose must be depressingly large to do any good. A course of the waters at Carlsbad, Contrexéville, or Ems may be recommended when practicable. No drug can be used as a solvent for oxalate of lime; but the patient should avoid rhubarb and other fruits, and be careful to live on a sparingly nutritious diet. Piperazin has been recently recommended for its solvent power over uric acid.

Moderate exercise, with attention to the condition of the skin, bowels, and digestive organs, are also important elements in preventive treatment.

In renal colic the first indication is to relieve the pain. Give opium or morphia, by the mouth or subcutaneously, to their full physiological effects. Hot baths and hot fomentations, or poultices to the loins may be applied. The patient must, of course, rest in bed. Subsequently our aim should be to help the speedy passage of the calculus. Belladonna is said to be useful by relaxing the spasm of the ureter. Give copious drinks of a warm and bland character, such as barley-water, toast-water. Should the agony continue intolerable, we may have to administer chloroform or ether.

The question of surgical interference will be influenced by the age and general health of the patient; the length of time he has suffered; the presence or absence of signs of amyloid degeneration; and also the condition of the opposite kidney. The stone may be excised, or it may be necessary to remove along with it an entirely disorganised kidney.

PARASITIC DISEASES OF THE KIDNEY

(1) **HYDATID CYST** is the most frequent form of parasitic disease, but still comparatively rare. The life history of the

¹ *R. Ammon*: Benzoatis gr. xx.; Spt. Chloroformi ℥xv.; Aquam ad ʒj. Misce.

parasite has been described under *Tænia echinococcus* (p. 300).

Symptoms.—Firstly, the patient complains of pain in the region of the kidney, and extending to the groin, testis, and thigh; then there appears a gradually-forming, globular, fluctuating tumour, resembling a hydronephrosis in site and anatomical relations. But the cyst is usually one of great tension, and as it enlarges it pushes aside every structure in relation therewith. Occasionally an hydatid thrill may be obtained by percussion, but we have never felt it ourselves.

Prognosis.—Usually favourable. (1) The cyst may rupture into the ureter, the bowel, the peritoneal cavity, the stomach, or even into the pleural cavity. If it discharge into the ureter, the presence of hooklets and daughter cysts, or shreds of cyst wall in the urine would afford a conclusive diagnosis. Occasionally a daughter cyst may be arrested in the ureter and set up symptoms simulating renal calculus. (2) It may suppurate and give rise to all the symptoms of renal abscess. The urine should still be searched for hooklets. (3) The parasite may die and the cyst shrivel, the contents being converted into a doughy mass which contains remnants of the cyst walls and hooklets.

Treatment.—Leave the tumour alone if it give rise to no pain or discomfort. Otherwise it may be punctured and drained, or injected. If it suppurate, the treatment is in all respects similar to that of renal abscess.

(2) *BILHARZIA HÆMATOBIA*.—A trematode worm; common at the Cape of Good Hope, in Egypt and the Nile Valley. The disease which it causes is therefore endemic in these parts. The mature worm infests water, and is supposed to enter the system by the alimentary canal; thence it migrates to the mesenteric or vesical veins, or to the kidney and ureter. Dr. John Harley thinks that the parasite may enter by the urethra during bathing. It is probable, as in nearly all of these internal parasites, that they have an intermediate host for their larval stage, and another host during their mature existence.

Symptoms.—Vary according to the part attacked. Should

the mesentery and bowel be involved, there would be a dysenteric form of diarrhœa, whilst affections of the kidney or ureter would give rise to pyelitis and abscess. Irritability of the bladder, attended by micturition of urine, at first clear, but containing blood at the end of the stream, with excess of mucus and of vesical epithelium, would point to affection of that organ. Occasionally hæmorrhage comes from the urethra after some slight exertion, and is not associated with micturition. In all cases the diagnosis, however, is incomplete until the ova are discovered in the urine or fæces, either free or embedded in sloughs of mucous membrane. The ova are elliptical bodies, pointed at one end, each containing embryos with long vibratile cilia. Beyond these signs, marked anæmia is a prominent symptom, and there is a general febrile condition somewhat resembling typhoid fever. In some cases, however, beyond slight and temporary hæmaturia the symptoms are quite trivial.

Treatment.—Kill the parasite if possible. Injections of quassia, iron, or turpentine may be applied to the bladder. Dr. John Harley recommends injections of iodide of potassium. Oil of male fern is of great advantage in order to rid the intestinal canal of the parasite. Beyond this, give tonics, such as iron and quinine, when the febrile disturbance has abated. The disease is said to be rarely fatal.

URÆMIA

Definition.—A term which is applied to certain nervous symptoms supervening on suppression of urine, or failure of the kidneys to eliminate the products of tissue-waste.

Causation.—The exciting cause is the circulation of urea in the blood. This may be due to any disease or condition by which the excretory functions of the kidney are arrested. It thus occurs in acute and chronic Bright's disease, in disorganisation of the kidneys by calculus, abscess, or new growth, and through the action of certain poisons.

Pathology.—The disease is due to arrest of the urinary excreta. The products of tissue-waste and tissue-change are

retained in the blood. The patient is then suffering from an *autogenetic* poison. Urea is found in the secretions of the various glands, except the kidneys, and in all dropsical effusions, and in the sweat.

Symptoms.—The symptoms vary considerably in different cases according to the secreting powers of the kidneys. Thus, in the acute nephritis which occurs after scarlet fever, or supervenes on the puerperal state, the onset is sudden and at times unexpected, but accompanied by almost complete arrest of the urinary excretion. In chronic renal disease, on the other hand, the typical symptoms are preceded by a preliminary headache, accompanied by drowsiness, nausea and vomiting, impaired vision, dyspnoea (worse at night), and a diminishing quantity of urine. The odour of the breath is said to be ammoniacal.

Subsequently one of two conditions supervenes, viz. the patient either becomes convulsed, or he passes into a state of coma.

The CONVULSIONS are like those of epilepsy in almost every respect. The patient falls to the ground insensible, his face is first pale, and then becomes livid. He has twitchings of the muscles, foaming at the mouth, and he often bites his tongue. The pulse is quickened, and there is usually some slight rise in the temperature. He may recover entirely from this attack ; or he may have a succession of similar seizures, with periods of semi-insensibility between—a condition which at once reminds us of the STATUS EPILEPTICUS. If he does not recover, he gradually passes into a state of coma, which is usually fatal. (See Eclampsia, p. 755.)

But COMA may be the first indication of the uræmic poisoning in another case. It may supervene gradually in thirty-six to forty-eight hours, or it may be sudden and complete, as in cerebral hæmorrhage.

The patient is quite insensible, nothing rouses him ; he breathes stertorously ; his respirations are slow, shallow, and often acquire a 'Cheyne-Stokes' character. The pupils are usually dilated, and the temperature slightly raised (100° about).

Diagnosis.—This is at once cleared up on passing a catheter into the bladder and finding little or no urine; and what urine there may be is highly albuminous. As distinguishing it from apoplexy, we often find uræmic convulsions without loss of consciousness, and uræmic coma without paralysis. In apoplexy these symptoms are combined.

Prognosis.—In acute uræmia the immediate prognosis is fairly favourable, especially if there is no reason to suspect advanced disease of the kidneys. The chronic form is more grave, but it is rare for death to occur after the initial seizure; usually it requires a succession of attacks to produce a fatal result.

Treatment.—During the convulsions the treatment should be the same as in epilepsy. Similarly in coma, the treatment in most respects is that which one would adopt in cerebral hæmorrhage.

One or two points, however, are of great importance. We should endeavour to lessen the arterial tension. This may be done by free venesection from the arm, or by wet cupping to the nape of the neck, or by leeches to the temples.

Subsequently encourage the bowels and the skin to activity, so that effete matters may be eliminated by these channels. A brisk purgative of croton oil, or of elaterium (gr. $\frac{1}{12}$), may be administered. Diaphoresis is quickest produced by the subcutaneous injection of nitrate of pilocarpine (gr. $\frac{1}{10}$) and by hot air baths.

Inhalations of chloroform may be required from time to time if the convulsions are severe or continuous. Experimental research points to the possibility of alleviation of the symptoms, or even cure, by the injection of the extract made from the kidneys of a sheep or other animal.

The diet should consist principally if not entirely of milk.

ISCHURIA (SUPPRESSION OF URINE)

Definition.—A term which is applied both to functional suppression and to retention of urine from obstruction.

Causation.—There are consequently two groups under

which cases may be classified, viz. : (a) Functional Suppression, and (b) Obstructive Suppression.

(a) FUNCTIONAL SUPPRESSION.—This occurs in a number of various conditions and diseases. It may supervene during an attack of acute Bright's disease, and during the course of some of the specific fevers, notably scarlet fever and diphtheria, of which Bright's disease is a complication. In cholera also it is a prominent symptom, not so much owing to any renal change, as to arrest of function of the kidney in common with other secretions. Suppression may also be a neurotic manifestation, as in hysteria and in shock. It may also be brought about by the actions of certain poisons, such as cantharides and turpentine. Finally, it may be produced by embolism or other form of obstruction existing in the aorta or in the renal arteries.

(b) OBSTRUCTIVE SUPPRESSION.—This is generally due to the existence of a calculus, or of new growth involving the pelvis of the kidney, or the ureter, bladder, or urethra. For the complete development of this form, it is necessary that the cause of the obstruction should implicate both ureters, else the opposite gland may carry on the functions of both. Obviously, therefore, those obstructions which exist in the bladder and the lower urinary tract produce the most marked results.

Symptoms.—In those conditions in which the urinary function is in abeyance, the symptoms vary somewhat in different cases. In cholera, for example, the condition seems to have little or no effect on the patient. In hysteria, again, the arrested urinary function may exist for days without causing constitutional disturbance beyond vomiting. In the majority of other cases, however, it is in itself a grave condition, speedily inducing exhaustion, the typhoid state, and death. If the kidneys again take on a healthy action, as they suddenly do in some instances, recovery is equally rapid.

In obstructive suppression the onset of symptoms is generally sudden. At first there is no marked inconvenience to the patient ; afterwards characteristic symptoms ensue. These are, headache, vertigo, vomiting, thirst, and muscular

twitchings. Subsequently the respirations become shallow, the pupils are contracted, the patient becomes drowsy and finally convulsed. But there is no dropsy and no rise of temperature. Death occurs in about a week from the onset of the obstruction; but complete recovery may take place on removal of the cause.

Treatment.—In suppression of function, all that we can hope to do is to alleviate symptoms by hot baths, and in some instances by purgatives. In most cases death occurs from the fever or other disease of which suppression is a complication.

In the obstructive form the main indication is to remove the cause, and consequently the treatment is more surgical than medical. In renal calculus Sir W. Roberts advocates kneading the abdomen in the region of the kidney and course of the ureter, with a view to dislodge the stone. The question of nephro-lithotomy may have to be considered.

In obstruction from malignant disease our treatment can only be palliative.

HÆMATURIA

Definition.—The presence of blood in the urine. The hæmorrhage may come from the kidney or from any part of the urinary tract.

Causation.—The causes may be (a) Local or (b) General.

(a) LOCAL.—(1) From the kidney, as a result of acute nephritis, calculus, new growth, parasites, injury, &c. The blood here is usually uniformly mixed with the urine, which hence becomes of a 'portery' tint. The presence of blood-casts is almost diagnostic. (2) From the pelvis, infundibula, or ureter, being the effects of new growth, calculus, parasites. The hæmorrhage is often profuse and periodical. (3) From the bladder, caused by inflammation, new growth, calculus, or parasites. The blood in such cases is frequently profuse, free, or clotted, and appears at the end of micturition. It is also intermixed with an excess of mucus or pus. (4) From prostate or urethra, owing to impacted calculus, stricture, injury, or ulceration. These causes are chiefly surgical. (5) Injuries to, or abscesses extending into, any part of the urinary tract.

(6) From the uterus during menstruation. (7) Vicarious menstruation, a very doubtful phenomenon.

(b) GENERAL.—(1) Poisons acting directly on the kidneys (turpentine, cantharides). (2) Specific fevers (notably typhus and small-pox). (3) General diseases in which hæmorrhage is a prominent symptom, *e.g.* purpura, scurvy. (4) Hæmophilia.

The diagnosis of the seat and cause of hæmorrhage will, however, in all cases depend on other collateral evidence and symptoms, in addition to the urinary signs. Such symptoms are referred to under the various diseases and conditions giving rise to hæmaturia. (See Calculus ; Malignant Disease ; Parasites of Kidney.)

In all cases careful palpation or exploration of the kidney, bladder, and urethra is necessary. The microscope and the endoscope will also in most cases yield valuable evidence of a negative or a positive character. It is necessary, however, not to mistake healthy renal or other epithelial cells for those of malignant new growth. And we should also be aware that the urine and its chemical constituents alter the appearances of the red discs considerably. They may be swollen, or more contracted than usual ; they also tend to lose their biconcavity of surfaces ; and their borders may be eaten out or crenated.

Treatment.—Absolute rest in bed is essential. The room should be cool. Iced drinks may be given. Avoid alcohol. Astringents may be administered, such as iron (the tincture of the perchloride is best), lead, gallic acid, and turpentine in small doses. The most potent drug, however, is ergot, either by the mouth, in the form of the tincture or liquid extract, or by subcutaneous injection. Digitalis and opium are also powerful adjuvants. Hæmorrhage from the bladder is best treated locally, by the application of cold to the perineal and hypogastric regions ; or by the injection into the bladder of iced water, or of diluted solutions of iron preparations (tincture of the perchloride, 1 in 30) ; or of tannic acid (1 in 20) ; or of hot water, to which may be added tincture of hamamelis (5j. to a pint), or calcium chloride (1 per cent. solution).

It is necessary that the bowels should be kept freely

moved. The question of removal of new growth or calculus must be discussed from a surgical standpoint.

PAROXYSMAL HÆMOGLOBINURIA (HÆMATINURIA)

Definition.—A disease characterised by sudden ague-like seizures, accompanied by the voiding of urine containing hæmatin, with apparently good health between the attacks.

Causation.—Obscure at present. From the periodicity of the attacks there is reason to associate them with some malarial taint. The direct exciting cause is often sudden chill or exposure to cold. It is said to be caused by taking glycerine in large doses, which has a solvent effect on the colouring matter of the blood. The cases are almost entirely limited to adult males who are subject to severe muscular exertion.

Pathology.—Not yet thoroughly established. During the attacks the kidneys are markedly congested, but there is no epithelial or other histological change. The red corpuscles of the blood, however, are destroyed, their hæmoglobin thus passing into the urine. The disease is probably related on the one hand with ague, and on the other with rheumatism.

Symptoms.—The attacks are paroxysmal. They are ushered in suddenly with rigors, severe lumbar pains, 'goose-skin,' retraction of testes, a tendency to yawn, vomiting, together with general malaise, and aching of limbs; in fact, all the symptoms which one might look for at the onset of an aguish attack; but the temperature of the body is sensibly lowered. The urine at the time of the attacks is bloody, contains much albumin, and deposits a grumous sediment. Microscopically, however, we fail to find any blood corpuscles, owing to their destruction and the solution of their contained hæmoglobin; but tube-casts, brown-coloured masses, and oxalates are found.

The spectroscope gives two bands in the green and yellow, and also occasionally a band in the red, showing the presence of hæmoglobin in the urine.

After a few hours there is a return of the normal tempe-

perature, or even a little febrility, and the attack gradually passes away, leaving the patient somewhat weak and exhausted. Subsequent seizures may occur at intervals varying from four or five hours to a week or more. The secondary attacks are a repetition of the former ones, but often in diminished severity, and they are, as a rule, dependent on sudden access of cold. The urine between the paroxysms will be found quite normal.

After many bouts a characteristic malarial, jaundiced, or anæmic complexion is acquired.

Treatment.—**PROPHYLACTIC.**—Avoid exposure to cold and damp. The body should be well clothed. Flannel garments are essential both in winter and summer.

CURATIVE.—During the attacks give ergot subcutaneously, together with copious warm non-alcoholic drinks. Subsequently iron and quinine, separately or combined, or arsenic, are indicated.

CHYLURIA

Definition.—A disorder characterised by the appearance of chyle and lymph in the urine, due to obstruction and rupture of the lymphatics.

Causation.—**Age.**—It occurs more frequently in adult life. **Sex.**—Women suffer in greater proportion than men. **Climate.**—Occasionally in this country cases of chyluria occur from obstruction of the thoracic duct, or of the lymphatic capillaries of the lower part of the body, as a result of tubercle or other new growth. That form of chyluria which is produced by parasitic invasion occurs most frequently, if not entirely, in tropical lands. The parasite, or exciting cause, is a nematode worm, the *Filaria sanguinis hominis*, which is nocturnal in its habits (see p. 304).

Pathology.—The lymph-vessels, especially those of the pelvis and lower part of the abdomen and legs, are obstructed at various points. As a result of this obstruction the lymphatic capillaries are dilated, either in their whole length below the obstruction, or locally, so as to form here and there pouch-like dilatations or vesicles which contain chyle. Cases

are recorded in which the thoracic duct was impervious at a certain level, with resultant enormous dilatation of the tube below. When the obstruction occurs in the lymphatics of the scrotum the disease is known as Lymph Scrotum. Elephantiasis is the name which is applied to a similar condition in the legs. Chyluria results when the obstruction takes place in the lymphatics of the bladder and urinary tract. The parasitic cause of the obstruction is probably not due to the presence of the mature worm, so much as to its embryos, which are too large to pass along some of the lymph-capillaries, and therefore play the part of emboli. The filaria itself easily travels through the capillaries. Chyluria here is only a manifestation of the parasitic invasion of the lymph-vessels of the bladder or urinary tract, which become dilated and ultimately rupture.

Symptoms.—The formation of cutaneous vesicles on the scrotum and the appearance of elephantiasis are sufficiently characteristic symptoms. But the most remarkable signs are presented by the urine. This, when first voided, is milky in appearance and in odour. It contains chyle, lymph, fibrin, and the normal constituents of urine, but the latter are in diminished quantities. Therefore it soon coagulates into a semi-solid or jelly-like consistence, which is translucent or opaque according as lymph or chyle predominates. After a time it again becomes liquid, and the chyle floats to the upper surface in molecular form, like cream, or fat. It also deposits leucocytes and blood, together with embryos of the parasite. This condition may be constant, or periodic only.

All these events are not inconsistent with apparent good health ; but sooner or later the patient emaciates ; he complains of obscure or dragging pains in the loins, hypogastrium, and perineum ; he has some slight fever, and becomes mentally depressed.

Diagnosis.—The diagnosis of the actual condition chyluria is easy. The scum which forms on the top of the urine is proved to be fat by its solubility in ether. The discovery of the parasite is of the utmost importance, since chyluria may occur from obstruction of the thoracic duct by tumour.

Prognosis. It is not necessarily a fatal disease. Patients

occasionally live a number of years ; the duration of life apparently depending on the degree and rapidity of the emaciation.

Treatment.—(a) PROPHYLACTIC.—Thoroughly cleanse all vegetables and fruits which are eaten in an uncooked condition, and boil all drinking-water. This latter precaution is at once obviously necessary when we consider the cycle of the parasite. A mosquito is supposed to be the intermediate host of the worm. After a debauch of human blood and the reception of the filaria into the insect's stomach, where the embryos further develop, it is supposed, on good grounds, that the mosquito in turn deposits the embryos in the water of rivers ; and thus they come to be received into the stomach and blood-vessels of human beings.

(b) CURATIVE.—Astringents have been advocated, especially gallic acid. Much more hopeful results should be anticipated from the application of astringent lotions to the bladder, such as a solution of nitrate of silver (gr. $\frac{1}{8}$ ad $\bar{\text{ss}}$ j.).

Other indications for treatment are to supply the waste of fat by giving a generous diet. Iron tonics will be required to correct the subsequent anæmia.

DISEASES OF THE NERVOUS SYSTEM

ANATOMY

The **BRAIN** consists of the two Cerebral Hemispheres and their basal ganglia, the Corpora Striata, and the Optic Thalami, the Corpora Quadrigemina, together with the Crura Cerebri, Pons Varolii, Medulla Oblongata, and Cerebellum.

The weight of the brain is about fifty ounces, and its proportion to the weight of the body is as one to fifty.

Cerebrum.—Each half of the cerebrum, when denuded of its membranes, presents for examination an external, an internal, and an inferior surface.

The external surface is marked by three most important fissures which indicate the boundaries of the various lobes. These fissures are known as the Sylvian, the Rolandic, and the External parieto-occipital.

(1) The Sylvian fissure is a wide gap dividing the external surface into two almost equal portions. It begins below at the anterior perforated space, and running upwards and backwards, separates the temporo-sphenoidal lobe from the rest of the brain; a precentral or vertical limb passes upwards from the main fissure soon after it appears on the external surface.

(2) The fissure of Rolando begins at a point in the great longitudinal fissure, about midway between the anterior and posterior extremity of the brain, and runs forwards and downwards to the angle formed by the vertical and horizontal limbs of the Sylvian fissure. It is recognised at once by its separating two parallel convolutions, viz. the ascending frontal and the ascending parietal. These are the only two

well-marked vertical, or rather oblique, gyri with a long sulcus intervening, and thus their identification is easy.

This fissure and its parallel convolutions are known as the Rolandic or motor area, since in this area are contained the cortical centres for the leg, arm (shoulder, elbow, wrist, fingers), mouth, jaws, pharynx, larynx, in this order from above downwards.

The centres for the muscles of the head and eyes are more anteriorly situated, viz. at the upper ends of the first and second frontal convolutions.

(3) The external parieto-occipital fissure is short and wide.

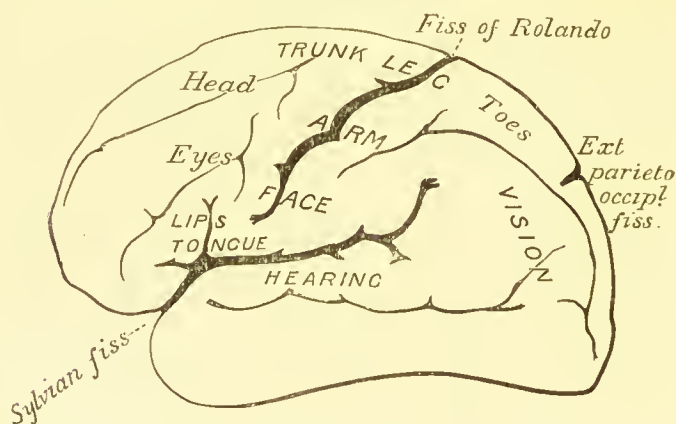


FIG. 22.—LEFT EXTERNAL SURFACE OF BRAIN

It is situated about midway between the superior end of the fissure of Rolando and the posterior angle of the cerebrum.

Thus for all practical purposes these three fissures divide the outer surface of the hemisphere into four lobes, viz. : the Frontal lobe, which extends from the anterior border of the brain to the fissure of Rolando ; the Parietal lobe, which is between the fissure of Rolando and the external parieto-occipital fissure ; the Occipital lobe, between the external parieto-occipital fissure and the posterior extremity of the brain ; and the Temporo-sphenoidal lobe, which is that part which is below the Sylvian fissure.

On the inner side of the hemisphere there are four primary

fissures, viz.: the Calloso-marginal, the Internal Parieto-occipital, the Calcarine, and the Dentate.

(1) The Calloso-marginal fissure begins at the anterior extremity of the corpus callosum, winds round and is parallel to this body, and finally finishes at the edge of the longitudinal fissure a short distance behind the fissure of Rolando, or about at that spot in the longitudinal fissure which would be struck by a line drawn vertically through the posterior border of the corpus callosum.

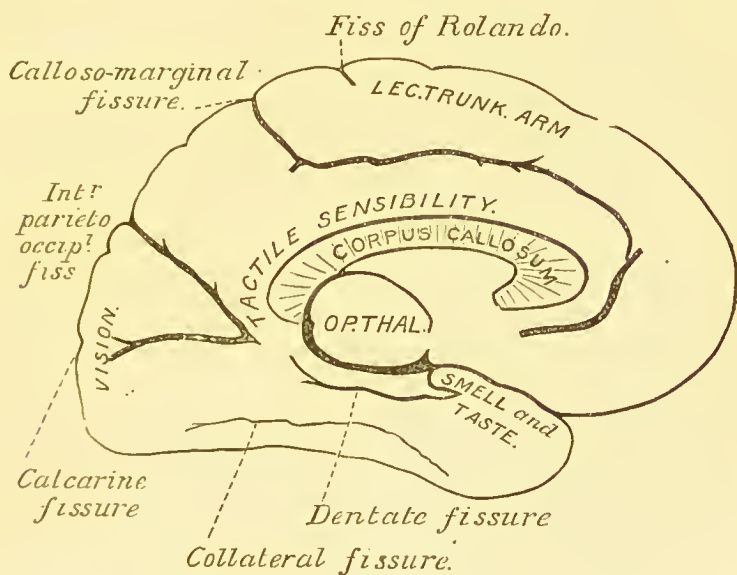


FIG. 23.—LEFT INTERNAL SURFACE OF BRAIN

(2) The Internal Parieto-occipital fissure commences at a point midway between the superior end of the fissure of Rolando and the posterior angle of the brain. It runs downwards and forwards to join the (3) Calcarine fissure at an angle of about 45° .

(4) The Dentate fissure begins at the posterior end of the corpus callosum and runs forward to the recurved hook of the uncinate convolution.

The Collateral fissure is again below this, with the hippocampal gyrus intervening.

As on the external surface of the brain, these internal fissures form boundary lines between lobules or convolutions.

The Quadrate lobule is between the upper limit of the calloso-marginal fissure and the internal parieto-occipital fissure.

The Cuneate lobule is between the internal parieto-occipital and the calcarine fissures.

The convolution between the corpus callosum and the calloso-marginal fissure is known as the gyrus fornicatus.

The Uncinate gyrus is immediately below the dentate fissure, and terminates anteriorly in a hook-like process, hence its name.

The *Corpus Striatum* contains three grey nuclei, the caudate, the lenticular, and the claustrum, which are separated from each other by white fibres. The caudate nucleus is in the floor of the lateral ventricle. The lenticular nucleus is more inferior and external, being isolated from the former by a band of white fibres, the internal capsule. The claustrum or *tenuiform* nucleus is, again, still more external, and is embedded in another layer of white fibres called the external capsule.

The *Optic Thalami*, *Corpora Geniculata*, and *Corpora Quadrigemina* receive fibres from the posterior and lateral aspects of the brain. Their various functions are, as yet, not fully determined. According to A. D. Waller, it is possible that the internal geniculate bodies and the testes may be the subcortical station of auditory sensation; and that the external geniculate body and testes are connected with the optic nerve.

The *Crura Cerebri* connect the hemispheres with the pons. On section, each crus presents two divisions: a ventral part called the crusta, the fibres of which transmit chiefly motor impulses; and a dorsal part, or tegmentum, the function of which is not yet definitely known. Some nerve cells, the locus niger, separate these portions, and the third nerve also comes to the surface at about the line of separation. The motor fibres of the crusta are arranged in special bundles for the tongue, lips, face, arm, leg, and trunk, in this order from within out-

wards. Thence they run through the pons, the majority (over 90 per cent.) decussating at the anterior pyramids.

The **Cerebellum** is connected above with the red nuclei of the tegment and optic thalami, laterally with the pons Varolii, and inferiorly with the medulla. It is composed of grey matter externally, and white matter internally, as in the cerebrum. It consists of a principal median ridge or lobe, the vermiform process, and two large lateral lobes. The function of this part of the nervous system is still undecided. Injuries on one side cause rotatory movements of the body towards the side of injury; and from experimental research and clinical investigation, the chief function which has so far been attributed to the cerebellum is that its 'action is an essential ingredient in the reflex guidance of muscular action' (Waller).

It is necessary, after these anatomical data, to trace the motor and sensory tracts from and to the brain. The *cerebral cortex*, or grey matter, is the brain proper; the white matter beneath conducts into it sensory impulses, and leads off from it, motor impulses.

At the base of the brain the white matter forms a conical sheet known as the internal capsule, which is the main channel of motion and sensation (Waller). This *internal capsule* is situated between the optic thalamus and caudate nucleus internally, and the lenticular nucleus externally. In vertical section it appears as a straight lamella, diverging slightly outwards, as it passes up to the corona radiata, and coursing downwards into the crusta. In

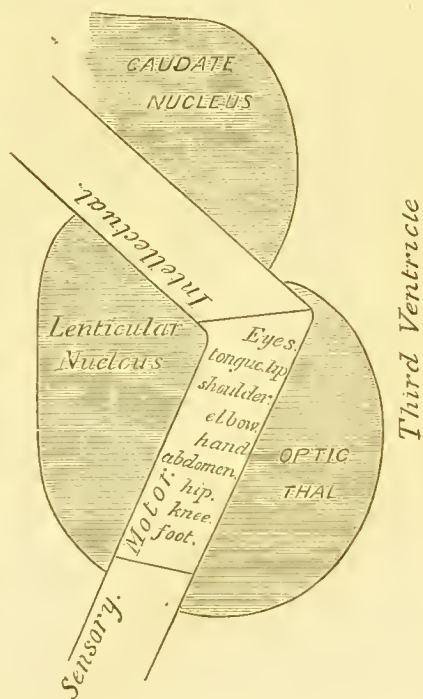


FIG. 24.—LEFT INTERNAL CAPSULE

horizontal section it presents a bend or genu, in front of this being the anterior third, which, diverging from the middle line, forms an angle of about 120° with the posterior two-thirds, which portion also diverges from the middle line. The

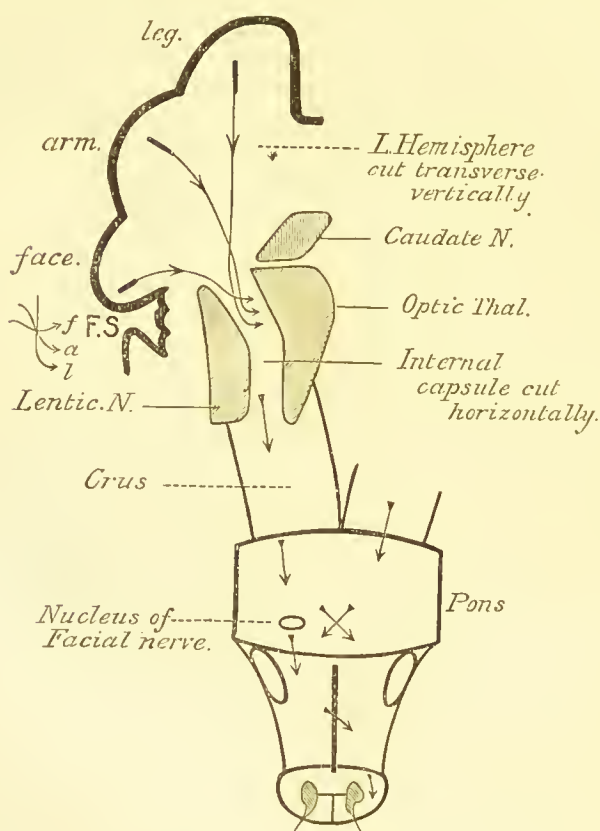


FIG. 25.—DIAGRAM TO SHOW THE COURSE OF THE MOTOR TRACT.
(Modified after Gowers)

anterior portion of the capsule consists of fibres which are associated with the intellectual faculties. The internal portion, or genu, is composed of fibres conveying motor impulses to the eyes, the tongue, and lips. The remaining fibres of the capsule are divided into three portions, the

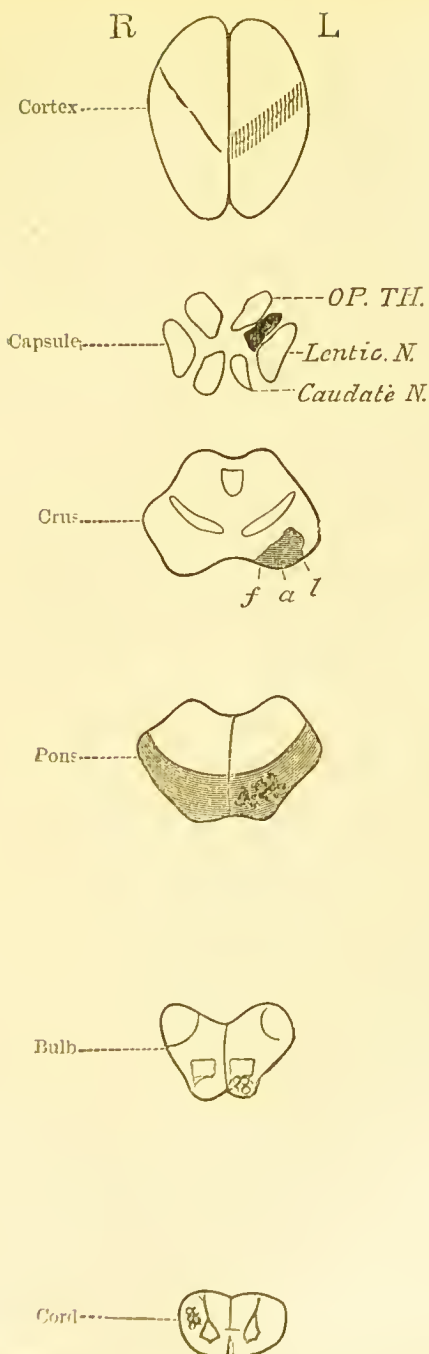


FIG. 26.—DIAGRAM TO SHOW COURSE OF DEGENERATION OF THE PYRAMIDAL TRACT. (After Gowers)

anterior two-thirds conveying impulses to the shoulder, elbow, hand, abdomen, hip, knee and foot in the order named, whilst the posterior third consists of sensory fibres, especially from the occipito-temporal region. The fibres for each limb are in separate and distinct bundles.

Thus the cortical fibres from the Rolandic area pass downwards into the corpus striatum, and occupy the anterior two-thirds of the posterior limb of the internal capsule ; and, further back, enter the crus cerebri and form its ventral layer or crusta. Thence the tract enters the pons, at the lower part of which it is in close communication with the nucleus of the corresponding seventh or facial nerve. At the lower border of the pons, the tract forms the anterior pyramid of the medulla. From this point the bulk of its fibres decussates across the middle line to form the crossed pyramidal tract of the cord, some few, however, continuing down on their own side as the direct pyramidal tract (see figs. 25 and 26).

The sensory tract is not so accurately defined. The posterior nerve roots, on entering the cord, are supposed to immediately cross to the opposite side ; thence they ascend to the posterior half of the medulla, and pons and crus ; but their exact position in these parts is not precisely known. Higher up, all the sensory fibres of the opposite side of the body are collected together in the posterior third of the internal capsule and ascend, some probably to the motor area, where muscular and tactile senses are located (Horsley), others to the uncinate convolution (Ferrier), others to the gyrus fornicatus (Schäfer).

Cranial Nerves.—The cranial nerves are arranged in twelve pairs, and make their exits through various foramina in the base of the skull.

For the superficial origins of these nerves, the student must consult some text-book of anatomy. The following diagram, however, will be of some assistance.

There are one or two points of importance which arise from the anatomical position of these nerves. It will be observed that the third pair of nerves are nearest together, and are therefore the most liable to be symmetrically involved by a single lesion in the median line of the brain. After these,

the sixth pair are nearest together, whilst the fifth are furthest apart. On the other hand, from their contiguity to each other, the sixth and seventh pairs, the seventh and eighth pairs, and the ninth, tenth, and eleventh pairs are more liable to be combined in a unilateral lesion. The deep origins are briefly discussed under 'Lesions of the Cranial Nerves.'

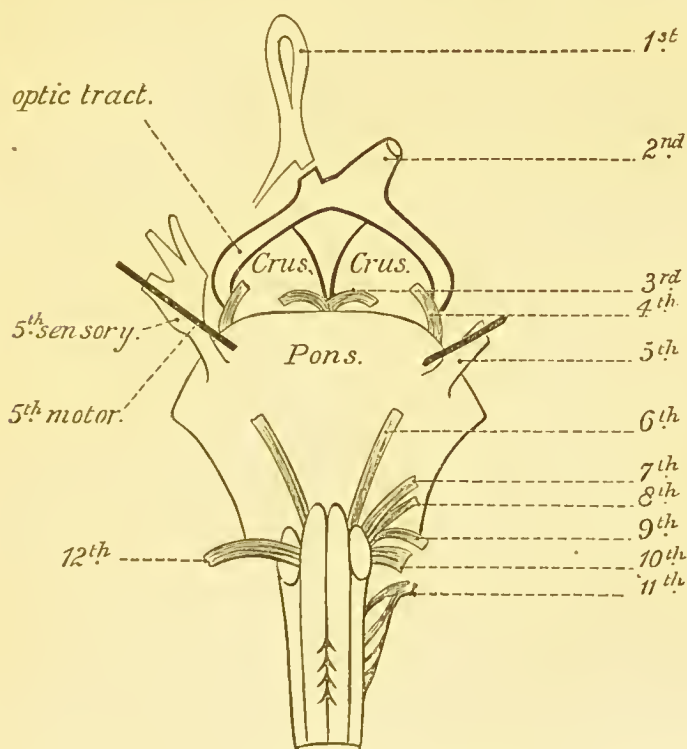


FIG. 27.—DIAGRAM TO SHOW SUPERFICIAL ORIGINS OF THE CRANIAL NERVES. (After A. D. Waller)

Cortical Centres of Special Senses.—The centre for *Smell* is situated in the uncinate gyrus. (Ferrier.)

The centre for *Vision* is situated in the cuneate lobule, and probably extends into the external surface of the occipital lobule. The centre for *word-vision* is in the angular gyrus, which is at the posterior extremity of the Sylvian fissure.

The centre for *Hearing* is represented in the first temporo-

sphenoidal convolution. That for *word-hearing* is probably in the upper edge of the same convolution.

The centre for *Taste* is at present undefined. It is possibly situated at the anterior end of the uncinate gyrus.

Effects of Lesions.—For diagnostic purposes, therefore, especially as regards the localisation of such diseases as cause gross lesions, the following points are important :

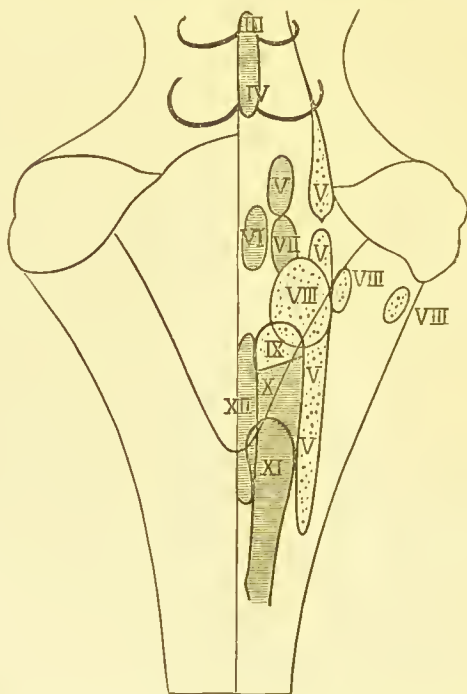


FIG. 28.—BULBAR NUCLEI. FLOOR OF FOURTH VENTRICLE, IMAGINED AS TRANSPARENT. MOTOR NUCLEI (INDICATED BY HORIZONTAL LINES): III, IV, VI, XII, V, VII, X, XI. SENSORY NUCLEI (INDICATED BY DOTS): V, VIII, IX. (After A. D. Waller)

Cortical lesions are associated with convulsions, which may be unilateral or bilateral, according as the injury is on one or both sides of the brain.

Lesion of the upper third (about) of the Rolandic area causes motor disturbances in the lower limb.

Lesion of the middle third (about) of the Rolandic area is associated with motor disturbances of the upper limb.

Lesion of the lower third (about) causes motor disturbances of the mouth, jaws, pharynx, and larynx.

Lesion of the Angular gyrus (Pli Courbe) probably causes complete mind-blindness.

Lesion of the Temporo-sphenoidal lobe in its upper convolution causes loss of hearing in the opposite ear.

A complete lesion of the internal capsule causes motor paralysis of the opposite side of the body. Cases may occur in which the lesion is so minute as to cause a monoplegia of (say) the opposite arm, or face ; or by its involving the sensory fibres only, cause hemianæsthesia, or loss of special senses of the opposite side. But, as a rule, in gross lesions all the fibres are equally involved.

Lesion of the Crus Cerebri causes paralysis of the face, arm, and leg of the opposite side, and of the eye on the same side as the lesion.

Lesion of the Corpora Quadrigemina usually implicates the nucleus of the third nerve, and hence causes oculo-motor palsy.

Lesion of the Pons, in the upper part, causes the same signs as lesion of the internal capsule, no cranial nerve being implicated. If in the lower part, besides causing hemiplegia of the opposite side, it would paralyse the fifth, sixth, and seventh nerves of the same side, and produce loss of conjugate deviation to the same side as the lesion. If the lesion be very extensive, so as to involve the fibres of the tegmentum, there would be, in addition, hemianæsthesia on the same side as the hemiplegia.

Lesions of the Medulla are, as a rule, so rapidly fatal that separate or characteristic palsies are seldom observed. A smaller lesion may cause paralysis of the hypoglossal and spinal accessory nerves.

Lesion of the Cerebellum causes vertigo, a reeling or ataxic gait, and vomiting. A localised symptom, such as deafness on the right side, combined with the ordinary cerebellar signs, points to a lesion of the right lobe. Paralysis of the fifth nerve, with other cerebellar symptoms, indicate disease of the middle peduncle.

The nuclei of the motor cranial nerves may be involved separately by degenerative changes. The nucleus of the third nerve, situated on the floor of the aqueduct of Sylvius, contains three centres, viz. for accommodation, reflex for light, and regulation of the external muscles of the eye, in this order from before backwards. Therefore a degenerative lesion of the anterior part would cause loss of power of accommodation and of reflex to light (ophthalmoplegia interna), whilst a lesion of the posterior part would cause oculo-motor paralysis (ophthalmoplegia externa). One or other, or all three, of these conditions may occur.

It is as well to note that probably the orbicularis palpebrarum receives its innervation, in part at least, from the third nerve nucleus, while the orbicularis oris may be innervated from the hypoglossal nucleus.

Lesion of the fifth nerve nucleus causes paralysis of the muscles of mastication, but probably not of taste.

When both nuclei of the sixth nerve are destroyed, the eyes converge. When the change is limited to one side only, it causes palsy of the external rectus of that side, associated with palsy of the opposite internal rectus. Thus the eyes are conjugately diverged from the side of the lesion.

Lesion of the seventh nucleus causes facial paralysis.

Lesion of the twelfth nucleus causes paralysis and atrophy of one half of the tongue ; but owing to the fact that there is usually an associated paralysis of the soft palate and of the vocal cord, it is possible that the muscles of these parts may receive some fibres from the twelfth nucleus.

Vascular Supply of the Brain.—The main arteries to the brain are the internal carotids and the vertebrals, these latter vessels uniting at the posterior border of the pons to form the basilar artery.

The internal carotid at the inner end of the Sylvian fissure divides into four branches, viz. :

(1) The anterior cerebral, which curves round the corpus callosum, supplying it, together with the optic chiasma, the olfactory bulb, the first frontal convolution, and the inner

aspect of the hemisphere as far back as the quadrate lobe. It is connected with its fellow by a short communicating branch which crosses almost immediately in front of the optic chiasma. The anterior cerebral therefore supplies the centres for the trunk and leg muscles (see fig. 23, p. 571).

(2) The middle cerebral runs in the Sylvian fissure, and supplies the corpus striatum, the second and third frontal, the ascending frontal and parietal convolutions, the middle lobe, the island of Reil, and the first and second temporal convolutions. It will thus be seen that this is the most important cerebral artery, supplying all the cortex except the first frontal convolution, and the lower parts of the occipital and of the temporo-sphenoidal lobe, and by its internal branches the greater part of the corpus striatum. One branch especially, the lenticulo-striate, is prone to hæmorrhage. Thus the main artery is responsible for the integrity of the greater part of the motor area, and also for the centres for speech, hearing, and vision (see fig. 22, p. 570).

(3) The posterior communicating runs directly backwards to join the posterior cerebral of the basilar.

(4) The anterior choroid is a small branch which, entering the descending horn of the lateral ventricle, is distributed to the hippocampus major and the velum interpositum.

The vertebral artery supplies the medulla and cord by means of its anterior and posterior spinal branches; and also gives off a posterior inferior cerebellar branch.

The basilar gives off an anterior cerebellar branch, branches to the pons, a superior cerebellar branch, and then divides into the right and left posterior cerebral arteries, with which the posterior communicating arteries join, as before stated, to complete the circle of Willis. The basilar is important in that it supplies the respiratory centre in the medulla, and hence obstruction of its early portion is much more alarming than of its termination.

The posterior cerebral artery supplies the under surface of the occipital lobe, the lower third of the temporo-sphenoidal lobe (including, therefore, the centres for smell and taste), and the hinder (sensory) part of the internal capsule.

One or two special features in the vascular supply of the brain are important.

The arteries of the base freely anastomose, forming the circle of Willis. The branches from this circle may be divided roughly into two sets, one supplying the cortex, the other the ganglia and internal parts. Both sets are really terminal arteries, and run to their area of distribution with little or no anastomoses. Consequently, when a branch is obstructed or ruptured, the area which it supplies is almost irretrievably damaged. The more minute internal vessels run into the brain-substance in sheaths or perivascular canals, an arrangement allowing of a certain degree of distension of arterioles without injuring the nervous structure. The arteries are *not* tortuous. The doctrine that tortuosity is a method of arresting the force of the current is not borne out by anatomical observation, so far as the cerebral arteries are concerned. Tortuosity of vessels is a feature which is associated with movements of parts.

The sinuses have no valves. The main blood-current is from before backwards. Coagulation is prone to occur in them, owing to the chordæ Willisii ; but from free anastomoses the effects of coagulation are less serious than one would suppose. The veins which are tributary to the sinuses enter these channels in a direction contrary to the main current. Venous anastomosis, on the other hand, is scanty, and hence thrombosis in these vessels is grave.

SYMPTOMATOLOGY

Nutritional Disturbances

In the course of many affections of the nervous system certain changes occur as sequels of the primary disease, in parts and organs other than the central nervous system. And although in many of such cases the first indication of change is rather of an inflammatory character, eventually evidences of atrophic changes are the more pronounced. At present physiologists are undecided whether the lesions to be mentioned are truly trophic in their nature, but suggest that in

many instances the lesions may be brought about by pressure, by injury, or by disturbances of the vaso-motor system of nerves.

Clinically, however, there is abundant evidence to show that disturbances of nutrition, as a sequel of structural disease of the brain, cord, or of the nerves issuing therefrom, may occur in the skin, muscles, bones, joints, viscera, and in the nerves themselves. The nerve lesions which cause these changes are for the most part irritative in character.

(1) SKIN.—Certainly bedsores form on the trunk and lower limbs in paraplegia due to spinal disease, and occasionally cases occur independently of the result of pressure. 'Glossy skin' (atrophy) supervening on cervical pachymeningitis, and herpes in the course of a cutaneous nerve, are also familiar examples of skin lesions.

(2) MUSCLES also undergo changes in nutrition: that is, they waste, and lose their electrical contractility, in destructive lesions of the anterior cornual cells. This is especially seen in progressive muscular atrophy and in infantile paralysis. A similar condition occurs in neuritis and in traumatic affections of the nerves which supply muscles; these muscular changes occurring independently of lesions of the motor cortex or spinal cord.

(3) BONES AND JOINTS.—Synovitis is a common complication of certain diseases of the brain, cord, or nerves; further, it is not always an isolated affection, since the muscles which move, and the adjoining skin which covers the joint, share a similar dystrophy, as though accentuating the anatomical axiom that the nerves which supply a joint also supply the muscles and skin in its neighbourhood. Amongst numerous examples of nutritional changes in joints and in bones may be mentioned the arthritis of the hands, which is common in prolonged lactation; the brittle character of bones found in locomotor ataxy; and the arrest of growth of, or withered arm, which is subsequent on disease of the anterior horn in the cervical bulb.

(4) VISCERA.—The urinary bladder has been quoted as the best example of a visceral lesion. That cystitis with ammoniacal urine is speedily found in paraplegic patients, in-

dependently of retention, is a clinical fact. Again, after division of the fifth nerve, the eyeball loses its sensitiveness, and ultimately inflames and ulcerates. But in both instances we are without actual proof of the existence of trophic nerves. The pathological fact, however, is in evidence, although the physiological reason is, as yet, not clear.

(5) NERVES.—When the connection of a nerve with its nutritional centre or cell is interrupted, the nerve itself also undergoes changes; it degenerates in that part which is beyond (in the direction of its impulses) its trophic centre. Thus, a lesion of the motor cortex, or of the internal capsule, causes degeneration which affects those nerves which, running down through the crus cerebri and pons, enter the crossed pyramidal tract of the cord. Also a lesion in the anterior cornual cells of the cord will cause degeneration of the motor fibres issuing therefrom, as well as of the muscles to which these fibres go. Similarly a lesion of the sensory root of a spinal nerve external to the ganglion causes degeneration of its fibres from below up to the ganglion; whilst a lesion internal to the ganglion causes degeneration of the central end of the root.

Hence the trophic centre for the motor tract is in the cortex of the brain; that for a spinal motor nerve is in the cells of the anterior horn; and that for a spinal sensory nerve is in the ganglion on the posterior root.

Sensory Disturbances

Sensation may be impaired, lost, rendered more acute, or otherwise modified in various nervous diseases.

(1) ANÆSTHESIA, or loss of sensation, is generally a cutaneous symptom, but not necessarily so; muscles and the organs of special sense may be similarly involved.

The patient often describes slight anæsthesia as a 'numbness,' or a 'dead feeling,' if it occur in the hands or feet. The degree and extent of anæsthesia should be estimated by the Faradic current, by stimulus of hot or cold sponges, or by other stimuli. Tactile sense is measured by an instrument known as the æsthesiometer, which consists of two sharp points of steel separated from each other on a graduated

bar. The perception of two distinct points obviously varies in different parts of the body. The tip of the tongue can detect two points only half a line apart; the skin over the middle of the spine only recognises two points which are thirty lines apart. These are the two extremes.

Anæsthesia occurs in lesions of the cord and brain when it is usually unilateral, in hysteria when it is unilateral or irregular in distribution, in general paralysis of the insane when it is universal, and in numerous other diseases and conditions.

(2) **HEMIANÆSTHESIA** is the term which is applied to loss of common and of special sensation on one side only of the body. It is usually due to disease of the opposite cerebral hemisphere, and when present it is generally associated with hemiplegia. But in hysteria, hemianæsthesia is often most profound, and is not necessarily accompanied by hemiplegia.

(3) **GIRDLE PAIN**.—This form, which is usually described by the sufferer as a ‘tight cord round the waist,’ is significant of inflammatory lesion in the cord; thus it may occur at various circumferences or levels of the body.

(4) **HYPERÆSTHESIA** consists of a painful sensation supervening on a slight stimulus, which ordinarily would be almost unnoticed. It is frequent in hysteria.

(5) **PARÆSTHESIA** or perverted sensations consist amongst others, of sensations of heat and cold, which are not in relation with abnormal or altered temperature; also of formications or ‘creepings,’ which occur in disease of the nerve trunks or of their peripheral endings. Intense itchings which occur in jaundice and some skin diseases would also be included under this category.

(6) **ANALGESIA** signifies insensibility to pain. The patient may perceive that a needle is being thrust into his skin, but it causes no pain. This phenomenon suggests the supposition that common sensation and pain travel up the cord by distinct routes.

(7) **MUSCULAR SENSE** is the mental estimation of the degree of contraction which is required by any set of muscles to perform certain work. In other words, the question arises can the patient distinguish a pound from an ounce? It is

needless, perhaps, to say that in this test the patient should not see the different weights.

Motor Disturbances

(1) PARALYSIS is loss of motor power. Diminution or incomplete loss of motion is known as paresis. Paralysis may be caused by damage to the (a) motor centres in the brain, or to the tracts below ; (b) to the cord ; (c) to the nerves issuing therefrom ; (d) it may be due to some disease or defect in the muscles themselves ; lastly, it may be (e) functional. From the above enumeration it will be seen that there are two sections of the motor path. The first, originating in the motor cortex, passes through the internal capsule, forming its greater part, through the crus and pons, then crosses over in the medulla to form the crossed pyramidal tract, finally terminating at the cells of the anterior cornua. The second section, starting from the anterior cornual cells, passes by the anterior nerve roots of the spinal nerves to the muscles.

(a) *Cerebral Paralysis*.—The lesions which give rise to this variety may be grouped as follows : (1) disease of the cerebral blood-vessels, such as atheroma, arteritis, thrombosis, and their resultant hæmorrhages ; (2) endocarditis, causing cerebral embolism and perhaps subsequent aneurysm ; (3) tumours, the most frequent being tubercle and gumma ; (4) abscess. The palsy is limited to one lateral half of the body (hemiplegia).

(b) *Spinal Paralysis*.—Lesions in the cord similar to those of the brain may occur. Caries of the vertebræ must also be added. The muscles which are supplied by nerves below the lesion are paralysed ; so that if the whole breadth of the cord is involved, all muscles below a transverse line drawn round the body are affected (paraplegia) ; and there is, in addition, loss of sensation. Occasionally the lesion affects only one half of the cord, and produces hemi-paraplegia. Besides loss of muscular power, incontinence or retention of urine, and loss of control over the anal sphincter, are characteristic symptoms.

(c) *Nerve Paralysis* may be occasioned by injury to the

spinal nerves at their exits from the vertebral column, by pressure of thickened meninges ; or by injury to the nerves in their courses, or at their peripheries. In such conditions the palsy is irregular, those muscles only becoming useless the nervous supply of which is cut off. Atrophy of such muscles is extremely rapid.

(d) *Muscle Paralysis*.—This form is usually due to direct injury or to want of use. It occurs, therefore, after some surgical operations, or consequent on fractures of limbs. For the most part the paralyses are temporary, and they affect one limb or one set of muscles only (monoplegia).

(e) *Functional Paralysis* occurs in hysteria and epilepsy. In hysteria the loss of power is apparently exaggerated, and migrates from one set of muscles or from one limb to another. The electrical reactions are not notably altered.

The effects of paralysis due to organic disease are seen in altered functions and qualities of the muscles. There is loss of movement ; loss of bulk (wasting), but in some cases increase of bulk ; loss of power and tonicity, and alterations in their electrical reactions. As a general rule we may state that paralysis due to cerebro-spinal lesion is attended by loss of power, followed by spasms, rigidities, and increased deep reflexes ; whilst paralysis dependent on lesion affecting the section between the cord and the muscles, is attended also by loss of power, but in addition by wastings and degenerative actions of these muscles and also by loss of deep reflexes.

(2) SPASM.—This term is applied to such contractions of muscle as are disproportionate to the amount of irritation or stimulus. The pathological irritation may occur at the great-nerve centres, or in the course of motor nerves ; a stimulus may be applied externally, to the skin for example ; and though, in itself, it is not necessarily pathological, it produces spasm in a disordered nervous system. Spasms are divided into two main groups, viz. (a) tonic and (b) clonic.

(a) *Tonic Spasm* is said to occur when muscular contraction of certain or regular intensity continues for an appreciable but indefinite period. It produces no movement when it affects antagonistic muscles ; when it affects one

group of muscles only, the limb or part is fixed at the extreme position which these muscles produce. Examples of tonic spasm are seen in tetanus and the early stage of epilepsy.

(b) *Clonic Spasm* consists of rapid muscular contractions alternating with relaxations; and hence various, and generally irregular, movements result. It is seen typically in the second stage of epilepsy, and in hysteria. Unilateral spasms are for the most part cerebral, and bilateral are spinal, in their origin.

(3) TREMOR is really a variety of clonic spasm, but the term is most frequently applied to movements of limbs or of other parts. Still tremors of muscle may occur, and they vary in degree from the fibrillar twitchings of muscular fibres, seen in progressive muscular atrophy, to the somewhat coarse movements of the limbs which occur in mercurial poisoning. Good examples of tremor occur in general paralysis of the insane (face and lips), in disseminated sclerosis (nystagmus), in paralysis agitans (pill-rolling action), and in mercurialism.

Tremors again are divided into two or three varieties—*e.g.*

(a) the *choreic* tremor is purposeless and irregular in character, and yet it tends to some definite muscular action. The movements are aggravated by attempts at voluntary co-ordinated movements; they cease during sleep, except in severe cases; and there is a period, more or less prolonged, of repose before each fresh jactitation. They convey to the spectator the idea that the motor cortex suddenly started a voluntary movement, and as suddenly withdrew the impulse (fig. 29).

(b) The *rhythmical* tremor occurs in typically disseminated sclerosis. The movements are small or large in different diseases, but they are all of much the same character, degree, and similitude (fig. 29).

(c) *Athetosis* is a form of convulsive tremor, characterised by choreiform movements of the fingers and toes, which cease during sleep. In adults it supervenes on hemiplegia, in which slight recovery has taken place, and is known as post-hemiplegic chorea. In children it is due to some congenital neurosis, or it is secondary to infantile paralysis, or to some injury to the brain shortly after birth.

(4) INCOORDINATION, or loss of the faculty of combining the action of muscles, is an important symptom. If the accord between those muscles which produce and those which control a movement is imperfect, incoordination results. There is no loss of power in the muscles, but, like an imperfectly trained crew, there is want of conjoint combination of power. The symptom is present in locomotor ataxy.

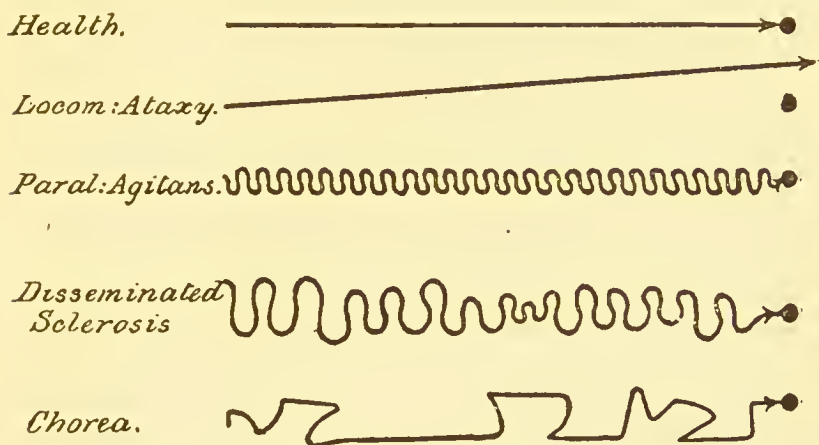


FIG. 29.—DIAGRAM TO REPRESENT TREMORS AND IRREGULARITIES OF MOVEMENT

(5) CONJUGATE DEVIATION.—This symptom is often observed in hemiplegia.

In health the movements of the two eyes are exactly equal and parallel for different directions of distant vision. In looking upwards the two superior recti, and in looking downwards the two inferior recti, act together. In lateral movements, however, the association of muscles is asymmetrical, the external rectus of one eye acting with the internal rectus of the other; just as in driving a pair of horses, each rein divides and is distributed to the check which is furthest from the pole in the one horse, and to the cheek which is nearest to the pole in the other horse. Bearing in mind this simile, the nose would represent the carriage-pole.

Now, on looking, say, to the right, impulses would pass from the left motor cortex (most probably) to the right external rectus and the left internal rectus, in which case the impulses

to the latter muscle must recross between the bulbar nucleus and the nerve's termination.

In hemiplegia the eyes and the head may be rotated to one or the other side, according to the different conditions of the lesion. Thus unilateral convulsions are accompanied by rotation of the head and eyes towards the convulsed side, *i.e.* away from the cerebral lesion. A discharging lesion, therefore, of the right cortex causes convulsions on the left side and rotation of the head and eyes to the left.

In destructive lesions, however, of the right cortex there is left hemiplegia with rotation of the eyes to the right. This is due to cessation of action along the left sixth nerve (external rectus) and right third nerve (internal rectus), the deviation of the eyes to the right being due to the unbalanced action of the muscles which pull to the right.

Similarly the head is rotated to the right by the left sterno-mastoid and the right inferior oblique and posterior recti muscles. Hence a sterno-mastoid and the opposite inferior oblique and recti muscles are conjugate muscles, and their conjugation is preserved as in the eyes; but the direction of the deviation varies according as the lesion is a *spasmodic* or a *paralytic* one. (See Waller's 'Physiology.')

REFLEXES

A. **Deep Reflexes** (*Tendon Reflexes*).—A term applied to the responses produced by suddenly tapping tendons or muscles, or by suddenly stretching them. The phenomena are not true reflexes, as Dr. A. D. Waller has shown, the response to the stimulus being too quick; but the term is a convenient one, and is in general use. Like all true reflex actions they depend on the integrity of the spinal cord at certain levels, as well as the integrity of the afferent and efferent nerves in connection therewith. In other words, the reflex arc must be unimpaired.

(a) The **KNEE-JERK** (*Westphal phenomenon*, *Patellar reflex*) is best known and most easily investigated. It should be present to a moderate degree in health. It is best elicited

by the patient sitting on the edge of a bed or table and allowing his legs to dangle listlessly. Or the operator can support the limb by placing his arm under the lower third of the patient's thigh. The extensor tendon of the knee is thus somewhat stretched, and the muscles rendered tonic.

Percussion should be made with a thin book, the rim of a stethoscope, or a specially made hammer, on the bare skin over the patella tendon, and not through garments. Some muscular exertion of the hands, such as pulling, grasping, &c., will increase the response.

The amount of knee-jerk in a normal condition can be clinically estimated by experience only. A certain degree is almost always present in health. Absence and exaggeration are both pathological signs.

The following table shows in what conditions the knee-jerk is absent and when it is increased.

<i>Absent</i>	<i>Increased</i>
1. Tabes dorsalis, involving the lumbar enlargement.	1. Primary lateral sclerosis.
2. Lesions of the anterior cornua, <i>e.g.</i> infantile paralysis.	2. Lateral sclerosis secondary to myelitis, compression of cord (tumour, Pott's disease, cerebral hæmorrhage).
3. Lesions (<i>e.g.</i> tumours, meningitis) involving the anterior and posterior nerve roots.	3. Disseminated sclerosis involving the motor tracts.
4. Various forms of peripheral neuritis. <i>e.g.</i> in alcoholic paralysis, diphtheria, diabetes, injury to nerves, cold.	4. Most forms of general neurosis (<i>e.g.</i> epilepsy, hysteria, neurasthenia, &c.).
5. Advanced pseudo-hypertrophic paralysis.	5. General paralysis of the insane.
6. In some cases of cerebellar tumour.	
7. In complete transverse lesion of the cord (Bastian).	
8. Usually in Friedreich's ataxy.	

(*b*) ANKLE CLONUS is the term which is applied to movements of the ankle due to clonic rhythmical contractions of the calf muscles. It is produced by suddenly and forcibly

extending (commonly called flexing) the foot on the leg, thus stretching the calf muscles and tendo Achillis. If this manœuvre fails, the contractions may be set going by a tap over the tibialis anticus. It is not present in health, and is therefore comparable to increased knee jerk, with which indeed it is generally associated.

It is elicited in degeneration of the lateral columns, irritation of, or pressure on, the cord (without lesion), and in hysterical paraplegia.

(c) TOE CLONUS is obtained by forcibly extending the toes.

(d) PATELLAR CLONUS.—On forcibly pulling down the patella and thus stretching the quadriceps tendon, rhythmical contractions of the muscle ensue. It occurs in lateral sclerosis.

(e) WRIST CLONUS is obtained by forcibly extending the wrist. It is seen in advanced hemiplegia with descending degeneration.

Other clonic or exaggerated movements in various parts of the body may be elicited in disease, such as the 'front tap contraction' of Gowers, or *periosteal reflex*, produced by tapping the inner surface of the tibia, which is followed by contractions of the quadriceps extensor muscle. Similarly there may be an increased and sudden contraction of the biceps on tapping the lower end of the radius (*bicipital reflex*), or of the triceps when the ulna is struck (*triceps reflex*).

A *jaw jerk* has occasionally been met with.

The significance of them all is the same as in ankle clonus. (See Suckling, *op. cit.*)

Myotatic contraction is a term suggested by Gowers for these various movements, as, according to him, they are *muscle reflexes*.

B. Superficial Reflexes (*Skin Reflexes*).—The phenomena included under this heading have a significance similar, or closely allied to, the deep reflexes. That is to say, their presence or their absence indicate the condition, healthy or otherwise, of certain levels or links of the spinal cord.

Some are present in health, others in disease only. They are produced by stimulations of the skin or mucous membranes of various parts of the body, which elicit certain

well-defined muscular contractions. In disease they are exaggerated in those parts which are under the influence of the cord below a lesion ; and, as showing their control by the cerebral cortex, they are absent from the paralysed side in hemiplegia.

The following table gives a list of cutaneous reflexes from below upwards :

Reflex	Point of Stimulation	Situation of Centre	Significance
1. Plantar . .	Irritating skin of soles	Extreme end of cord	Usual in health
2. Gluteal . .	Irritating skin of buttocks	Origin of 4th and 5th lumbar nerves	Rare in health
3. Cremasterie .	Irritating skin of inner side of thighs	Origin of 1st and 2nd lumbar nerves	Usual in health ; best marked in boys on account of the newly-formed cremaster
4. Abdominal .	Irritating skin of abdomen in line of nipples	Origin of 8th to 12th dorsal nerves	Frequently absent
5. Epigastrie .	Irritating skin of chest in 5th and 6th spaces	Origin of 4th to 6th dorsal nerves	May be absent in health
6. Erector Spinæ	Irritating skin from scapula to crest of ilium	Origin of all the dorsal nerves	Rare in health ; frequent in wasting diseases
7. Interscapular	Irritating skin between scapulæ	Origin of 6th cervical to 3rd dorsal nerves	Rare in health
8. Palmar . .	Palms of hands . .	Cervical bulb . .	Only in infants
9. Cranial : Conjunctival	Sclerotic, or inner surface of eyelid	Medulla . . .	Absent in disease of fifth nerve only
Iris (to light)	Pupil	Anterior portion of oculomotor nucleus	Absent in disease only
Palate . . .	Soft palate and uvula	Medulla . . .	Do.
Nasal (sneezing) . .	Naso-respiratory passages	Medulla . . .	Do.

There are other reflexes connected with vital, or at least important, functions, such as respiratory, cardio-inhibitory, vesical, defæcatory, sexual, the absence of which are only revealed by disease of the medulla or of the lumbar cord.

Some of the above reflexes, as will be seen, are absent in healthy states of the cord ; and, even in those which are indicative of disease, the morbid condition may not necessarily reside in the cord, but may involve the motor or

sensory nerves at some point away from the spinal canal. But the presence of reflexes immediately above and below one which is found to be absent, is strong evidence of disease at that cord centre, or in the loop with which it is in association.

Electrical Reactions

Muscles in health respond to the action of electricity whether applied to the nerves supplying them (motor points) or directly to the muscular fibres themselves. The response to such stimulus, when applied to a nerve, is an evidence of the integrity of its muscular terminations ; and since the nutrition of a muscle depends on its healthy connections with its nerve, we are enabled to estimate, not only the condition of the nerve, but also the amount of trophic change which has occurred in the muscle itself as a result of nerve injury. The intensity of the stimulus required to cause a muscular contraction varies, therefore, considerably according to the state of the nerve, and also of its muscular periphery ; and so long as the muscle is not entirely withered, it will, although palsied, respond to an electric current if sufficiently strong.

Two forms of electricity are in general use in medicine, faradism and galvanism.

A. The *Faradic*, or induced current, derived from an induction coil, consists of a series of shocks in rapid succession, so as to induce a tetanic condition of muscle. The current flows alternately in both directions, but causes muscular contraction only by its action on a motor nerve, either in its course or at its motor endings. Consequently, it produces no results when the motor nerve is destroyed.

B. The *Galvanic*, or continuous current, produces a muscular reaction only at 'making' and 'breaking' contact—when the current begins to pass, and the instant at which it ceases—but not during the flow. The current flows in one direction only, from anode (+) to kathode (—), and hence the current is called *descending* when the anode is near the centre and the kathode on the muscle, and *ascending* when the anode is on the muscle and the kathode nearer the centre.

The current being of comparatively low tension, it has only slight influence in causing muscular contraction.¹

Electricity is employed in medicine for two purposes, viz : as an aid to *Diagnosis* and as a method of *Treatment*.

(I) **Diagnosis**.—As a means of diagnosis both faradism and galvanism are used.

Faradism produces muscular contraction only through the nerve trunks or their endings in muscles, as previously stated. Given a healthy condition of these factors, a contraction of muscle occurs, the intensity of which varies according to the strength of the current used. This contraction may be practically regarded as a mild tetanus.

Should the nerve be degenerated, either from destruction of its peripheral endings, as in peripheral neuritis, or from disease of its spinal centre, as in acute anterior polio-myelitis the faradic reaction is diminished or lost, according to the amount of the lesion. Loss of faradic excitability is one of the factors of the 'reaction of degeneration.'

The galvanic current produces definite quantitative and qualitative reactions. In health the quantity of the reaction, or muscular contraction, may be described as short, sharp, and sudden. In disease this reaction may be simply increased (rare), or simply decreased, as in old-standing hemiplegia.

The quality of the reaction depends upon the relative position of the poles and the strength of the current. Hence, as there are two poles, and muscular contractions occur at making and breaking (opening and closing) of circuit, there are four forms of contraction. Thus, with

(a) The kathode or — pole on the muscle and anode or + pole on a neutral point, and a minimum current, a contraction occurs when the circuit is closed, or, represented symbolically, K.C.C.

(b) On greatly increasing the number of elements a contraction occurs when the circuit is opened, or K.O.C.

(c) With the anode on the muscle a contraction occurs in opening the circuit, or A.O.C.

¹ For a description of the apparatus for conducting, interrupting, regulating, and applying the electric force, the student should refer to one or other of the numerous text-books on electricity.

(d) A contraction occurs with the anode on the muscle on closing the current, or A.C.C.

The order of these reactions on increasing the current is K.C.C., A.C.C., A.O.C., K.O.C., the first requiring only a small number of galvanic cells; the last is only elicited by the strongest currents, which at times cannot even be tolerated.

In disease, when a peripheral nerve or its spinal centre is destroyed, a well-marked alteration in the above order of reactions takes place. This qualitative alteration constitutes the second factor in the 'reaction of degeneration.'

In order to appreciate the diagnostic value of electrical reactions, it is essential to remember the trophic value of the cells of the bulbo-spinal centres. Lesions above the centres, if followed by any electrical alteration at all, are associated with simple diminution of electrical excitability, as, for example, in old hemiplegia. Lesions of, and below, these centres cause secondary or Wallerian degeneration of the nerve fibres and degenerative wasting of the muscles which they supply, followed by the 'reaction of degeneration,' as, for example, in acute anterior polio-myelitis, and in all forms of peripheral neuritis.

The '*Reaction of Degeneration*' (R.D.), therefore, is a condition which ensues on lesions of motor nerves or of their nuclei. The nerves lose the excitability both to faradic and galvanic currents. The muscles which are supplied by these nerves atrophy, their response to the faradic current, and also to a less extent to the galvanic current, is diminished. This is followed by entire loss of response to faradism, whilst their excitability to galvanism is increased, *i.e.* the contractions which occur are slow and prolonged, and result from fewer cells.

Finally, galvanic irritability is lost, and muscular action is beyond recall. Thus, qualitative or polar changes occur, and instead of the normal order of excitability, as described above, a well-marked alteration in the order of reaction takes place, and A.C.C. may equal or exceed K.C.C., and K.O.C. may equal or be in excess of A.O.C.

This alteration may perhaps be more easily remembered when the reactions are placed in tabular form, thus :

Normal	Qualitative changes
K.C.C.	A.C.C.
A.C.C.	K.C.C.
A.O.C.	K.O.C.
K.O.C.	A.O.C.

(II) **Treatment.**—As a means of treatment electricity is used in paralysis, in spasm, in neuralgia, and (surgically) for galvano-puncture and galvano-cautery.

In paralysis, electricity acts chiefly as a stimulant by influencing the nutrition of the nerves and muscles which are involved. Our chief object here is to keep up the tone of the muscles, and, by preventing their atrophy, to preserve them in a healthy state until the natural nerve-currents are restored.

In spasm and in neuralgia electricity chiefly acts as a sedative agent. It is especially useful in some forms of painful spasm of the stomach, bowel, urinary bladder, and other hollow viscera. In neuralgia it may act through the imagination of the sufferer, or produce good results by diverting his mind from a previous torture. It also appears to be useful as a counter-irritant in sciatica, lumbago, and rheumatism. Certain rules should be observed in applying electricity as a therapeutic agent in paralysis.

(1) Apply faradism or galvanism according to the response which is obtained. An affected muscle which responds to faradism requires this form of electricity and not galvanism.

(2) Apply only the weakest current which is required to produce the desired effect.

(3) Diminish the current as improvement takes place.

(4) Each sitting should not exceed fifteen minutes, and during this time each affected muscle should be stimulated in turn, or each painful spot should be brought under the influence of the current.

(5) In using galvanism the kathode should not remain long on one spot.

(6) When volitional function and museular irritability are both lost it is useless to continue electrical treatment.

Galvano-puncture has been advocated in the treatment of hydatid cyst and in thoracic aneurysm. So far as the former is concerned, it would appear quite as easy to apply the ordinary hypodermic syringe, and quite as efficacious. Nor, in the few cases in which we have seen it applied in aneurysm, has it appeared to confer any benefit, but rather hastened the death of the patient. Whenever electricity is used in aneurysm a number of cells of low electro-motive power only should be used. (Poore.)

The galvano-cautery is employed almost entirely in surgical practice, and need not be considered here.

DISEASES OF THE BRAIN

SIMPLE MENINGITIS

Definition.—Inflammation of the membranes of the brain.

Causation.—(a) PREDISPOSING CAUSES.—*Age.*—Meningitis occurs most frequently in young children, but this preponderance is due to the frequency of tubercular meningitis, which forms the subject of a succeeding chapter. Those cases, however, which are secondary to injury and to general diseases, occur mostly in male adults between twenty and forty-five. *Previous diseases.*—As would be expected, all diseases which produce an active hyperæmia of the brain act as predisposing causes. It may thus be secondary to *specific fevers*, or it may occur as a complication of *acute rheumatism*; it is a condition often attending the chronic stages of *alcoholism*, and it is found to accompany *chronic Bright's disease*, and *tertiary syphilis*. It may occur epidemically as a form of specific disease (*cerebro-spinal meningitis*, p. 164).

(b) EXCITING CAUSE.—In most instances there is some direct exciting factor, such as inflammatory extensions from the mastoid cells, or internal ear, or nasal cavities, or invasion of suppurative disease from the cerebral sinuses. Meningitis may also be directly caused by blows or falls on the head, exposure to the sun's rays, or by prolonged mental exertion. It is probable, however, in the two latter instances, that meningitis does not occur without the influence of some one or other of the above predisposing conditions.

Pathology.—The post-mortem appearances are, as a rule, confined to the vertex of the skull, although after a while the

inflammatory processes may extend to the base, chiefly by extensions along the courses of the larger arteries. Inflammation of the dura mater is almost invariably caused by direct injury, or by extensions of inflammation from the ear, mastoid process, or the sinuses. In the idiopathic form the pathological changes are limited to, or accentuated in, the pia mater and arachnoid (leptomeningitis). The pia mater is at first engorged, and the surface of the brain dry and sticky; then the membranes become thickened and opaque, and effusion of serum occurs into the arachnoid cavity and into the sub-arachnoid space. Occasionally the effusion becomes purulent, the pus forming irregular patches on the surface of the brain by reason of its extension between the meshes of the pia mater, or it is arranged in streaks along the great fissures or following the courses of the large arteries. As a result of the inflammatory process, the pia mater is adherent to the convolutions, which become torn when an attempt is made to separate them. If the inflammation extends to the brain substance, as indeed it usually does, the convolutions are flattened and somewhat softened. On section they are seen to be engorged with blood, and show numerous vascular puncta and small extravasations. The pia mater in the ventricles (*velum interpositum*) may also present signs of inflammatory extension through the transverse fissure, with a result that the ventricles are filled with clear serum, or with a turbid purulent fluid similar to that found on the cerebral surfaces.

It is only necessary to add that the post-mortem appearances in all cases do not coincide with the above description, but that they will vary according to the stage at which death occurred, and also according to the intensity of the inflammation.

Symptoms.—In a well-defined case there are three well-marked stages, viz. *invasion*, *excitement*, and *collapse*.

The stage of *invasion* is usually sudden. It may be preceded by a general malaise, with defective appetite and perhaps some ringing in the ears, or other subjective symptom, but it is generally ushered in abruptly by fever, quick pulse, intense headache, vomiting, a flushed face, injected conjunc-

tivæ and rigors. The duration of this stage is uncertain ; it may be very brief, lasting an hour or two only.

The stage of *excitement* is characterised by aggravation of the above symptoms, together with others denoting nerve irritation. The pulse quickens (120) and becomes thin and hard, the temperature rises considerably (105°), there is great sensitiveness to light and sound, the eyelids are forcibly closed, the eyebrows knitted, the pupils contracted and fixed, and there may be spasmodic squint. The patient, at first peevish, restless, and sleepless, soon becomes delirious, the delirium frequently being of the wild or noisy type ; vomiting continues and is quite independent of food. Double optic neuritis is generally seen on ophthalmoscopic examination. The symptoms of this stage point to irritation of the cranial nerves.

After a variable time this stage is succeeded by that of *collapse*. The patient cannot be roused, he appears to have lost all sense of hearing and of sight, delirium abates or ceases entirely, and is replaced by stupor or coma ; the patient picks at the bedclothes (carphology), muscular prostration is shown by subsultus and relaxation of the sphincters ; occasionally rigidity of muscles takes place, either in general, or perhaps limited to one extremity ; his pupils are dilated and fixed ; the pulse becomes irregular, feeble, and thready, and the patient dies apparently from exhaustion. The symptoms of this stage are those produced by paralysis of cranial nerves, followed by coma. In some cases there is a variation as regards these symptoms. Occasionally the first and second stages are omitted, the disease commencing with convulsions, with coma speedily following.

Pain (headache) is usually present throughout the course of the disease, or until coma supervenes, but it is liable to remissions and exacerbations. The pain is generally so severe as to cause the patient to cry out, or moan, or roll his head about on the pillow.

Vomiting also is an early symptom and continues through the illness, or at least until coma comes on. It occurs independently of food, and is caused by the cerebral irritation ; it

frequently alternates with the paroxysms of headache, which it appears at times to relieve. It is not, as a rule, checked by any medicines.

Delirium is usually a late symptom. It is of the wild or violent type, restraint being required. The patient talks incessantly, and always requires careful watching, and his special senses are dull or perverted.

Convulsions more frequently occur in children, and may, but by no means always, replace delirium, or at least appear to represent the delirious stage in the adult. Occasionally they occur early in the disease. They involve the muscles of the face, limbs, or body, or they select special parts, such as one limb, or the tongue, or the eye muscles.

Rigidity is a late symptom; we may find it causing retraction of the head and various forms of squint. Before death occurs the rigidities, if present, usually relax, but leave the muscles quite limp and palsied.

In addition to the above, meningitis, when it affects the base of the brain, is accompanied by symptoms pointing, in the first place, to irritation of the cranial nerves, and subsequently to their paralysis.

Prognosis.—Unfavourable. Few cases recover; but recovery is more likely to take place in secondary meningitis than in those cases in which it appears to be the initial disease. Death may occur within forty-eight hours, but most commonly the fatal period is between the fourth and seventh days.

Diagnosis.—Simple meningitis has certain symptoms in common with tubercular meningitis, delirium tremens, hysteria, and typhoid fever.

The diagnostic points distinguishing simple meningitis from the tubercular form are given in a succeeding chapter.

In *delirium tremens* we find suspicions and delusions. The delirium is also of the 'busy' type, or the patient gives one the idea during his wanderings of terror or cowardice. Headache also is not present, and there is a previous history of alcoholic excess.

From *hysteria* the diagnosis is not always easy. The

shoutings or peevishness and the rigidities are all seen in hysteria, but there is no fever and no optic neuritis.

Typhoid fever would be recognised by the distended abdomen, the rash, and the characteristic stools. The disease also lasts longer; its temperature curve is characteristic, and neither vomiting, headache, nor delirium are urgent symptoms, at least during the first ten days; nor is the fever accompanied by muscular rigidities. The presence of otorrhœa, disease of the mastoid cells, or injuries to the cerebral sinuses, would, if present prior to a sudden attack of delirium, always lead us to suspect the onset of meningitis.

Treatment.—It is a disease in which something must be done speedily, and at the onset. Depletion is necessary, unless there be symptoms of exhaustion to forbid such measures. Apply three or four leeches to the temples, or bleed freely from the arm. Also give a quick, active purgative; calomel is best (gr. ij. to v.), and it may be repeated every other day.

Apply ice-bags, evaporating lotions, or cold douches to the shaved scalp. A hot bath acts as a sedative and is thus beneficial, and we may apply the ice-bags to the head at the same time.

Internally, iodide of potassium is always a safe remedy, even in cases other than syphilitic. Bromides help to control the delirium, but opiates are, of course, injurious. Aconite, in small frequent doses, may be tried with a view to arrest the febrile and inflammatory condition, but it is no use continuing it unless it speedily produces good results.¹ It is only necessary to point out that ear disease, or other causal suppurative affections of cranial bones or sinuses, demand local surgical treatment.

The patient requires careful nursing and vigilant watching to prevent his injuring himself during delirium. The room should be darkened and kept cool and well ventilated. Retention of urine may require the use of a catheter.

¹ R. Ammonii Bromidi gr. v.; Tinct: Aconiti ℥v.; Syrup Aurantii ℥x.; Aquam ad ʒij. Misce.

The diet should be scanty, but nutritious. Milk and eggs appear to be most suitable.

TUBERCULAR MENINGITIS

Definition.—Inflammation of the membranes of the brain, but especially of the pia mater, due to tubercular invasion, and usually running an acute course.

Causation.—(a) **PREDISPOSING CAUSES.**—*Age.*—A disease of early life ; but it may occur at any period, though with decreasing frequency after the second decade. *Sex.*—It occurs more frequently in boys than in girls. On examining statistics we find certainly a preponderance in males ; and as in a large percentage of these cases the meningitis appeared to have commenced acutely after some blow or injury to the head, it would appear to be probable that a tubercular deposit in the brain may lie dormant until roused to activity by some of the common injuries which boys are liable to, but from which girls escape.

(b) **EXCITING CAUSE.**—A tubercular invasion of the cerebral membranes. The meningitis is in most cases only a part of the general tubercular disease ; yet, although it is usually associated with a general tubercular invasion of the whole body, the lesion may be confined to the pia mater, just as we may meet with a tubercular joint only in another case, or caseous glands in a third, or a tubercular deposit in one lung only in a fourth instance.

Pathology.—The most marked pathological changes occur at the base of the brain. There are found signs of acute basilar meningitis, together with tubercles in various stages of growth. The pia mater appears most affected, lymph being poured out between its meshes, and producing a dull or sticky appearance of the structures at the base. The effused lymph is specially evident in the interpeduncular space, on the upper surface of the cerebellum, and along the Sylvian fissure. The tubercles are found either in intimate connection with the smaller arteries, or in the perivascular sheaths. They vary in size, some being as large as small

shot, others requiring the aid of a lens before they can be seen ; they are thus easily overlooked. They seem especially to congregate in the neighbourhood of the circle of Willis, along the course of the middle cerebral artery, and in the sulci. Their numbers do not necessarily bear any relation to the amount of lymph and other inflammatory products. They are usually grey in colour, as death occurs before there has been time for them to caseate. The ventricles are commonly distended by clear or slightly turbid serum, by which their shape and boundary walls are altered, the neighbouring sulci pushed out, and the convolutions flattened.

As tubercular meningitis is not usually a primary lesion, we should expect to find deposits in the lungs, peritoneum, spleen, or other situations.

Symptoms.—In a typical case, in a child, there are usually three stages of the disease, viz.: (1) STAGE OF INVASION OR EXCITEMENT ; (2) STAGE OF COMPRESSION ; and (3) STAGE OF PARALYSIS OR DEPRESSION.

(1) STAGE OF INVASION OR EXCITEMENT.—This stage cannot be said to have occurred until the typical symptoms are developed. It will, however, have frequently been noticed that for some weeks prior to this stage the child may have been listless and irritable ; that it has had a capricious appetite and lost flesh ; that the bowels have been irregular, diarrhœa alternating with periods of constipation. Frequently these symptoms, however, are overlooked, or attributed to some trivial disorder of the gastro-intestinal canal.

Then there supervenes a sudden access of fever, with severe headache, a quick pulse (120), constipation, and vomiting. Convulsions may or may not occur. The child is irritable, often refusing to be moved or examined ; there is photophobia, with occasional squints, or double vision.

(2) STAGE OF COMPRESSION.—In this stage the temperature falls somewhat, and the pulse becomes slower and intermittent. The mental condition is more obtuse, the headache continues, the head being rolled from side to side or retracted ; the child has tonic contractions or twitchings of the facial or other muscles ; spasmodic squints may occur, and the

ophthalmoscope reveals double optic neuritis, and probably choroidal tubercles. The bowels continue constipated; the belly walls are flattened or even concave ('boat-shaped'). Vomiting is still obstinate, and is in no way related to food. Irritability of temper is replaced by somnolence in the daytime, and by restlessness or delirium at night. A peculiar cry, resembling a kid's bleat, is frequently heard at this period.

(3) In the PARALYTIC OR DEPRESSED STAGE the pulse continues slow and irregular; the breathing is also slow, sighing and gasping, or it presents the irregular character known as the 'Cheyne Stokes' type. Constipation continues, but vomiting is often absent. The abdomen is more retracted, and the skin dry, harsh, and furfuraceous. The mental faculties are depressed; the patient lies on his back and is roused with difficulty. As the end approaches, convulsions with delirium ensue; the pupils dilate, but are insensible to light; and death occurs in coma.

It must not be supposed, however, that in all cases the above stages and symptoms are definitely marked. In some individuals there is merely a period of febrile invasion with intense headache and constant vomiting, which is speedily followed by convulsions and coma. In others certain characteristic symptoms predominate, whilst some which are equally diagnostic may be absent. The following symptoms, however, will generally be found at one or other period of the illness, and often persist from beginning to end.

Headache.—Always severe from the onset, causing the child to moan, or shout, and utter the characteristic cry or whine. It apparently ceases when delirium supervenes. Double optic neuritis may be observed from the first. It is almost invariably present in the later stages.

Vomiting, as in simple meningitis, comes on at all times. It is purely sympathetic, and is not related to food, nor is it preceded by nausea.

Retracted Abdomen is most marked as the disease advances. The skin generally is harsh, and not infrequently a scratch from a thumb-nail or any sharp instrument leaves a more or less red elevated mark or wheal, which lasts for some

time (*tache cérébrale*). Much importance has been laid on this symptom of vaso-motor paralysis ; but it is by no means constant or diagnostic.

Pulse.—At first quick and febrile ; it becomes slow, irregular, and intermittent after the first stage, from irritation of the pneumogastric nerves. This symptom with accompanying fever is an almost sure sign of meningitis.

Respiration is also of a febrile character at first, being quick and shallow. The sighing, and irregular character supervene later on.

Paralyses occur in the final stage. They are peculiar in that they are liable to come and go. To-day one arm may be palsied, and to-morrow some power will appear to have returned, whilst a leg or other part is involved.

Eyes.—Squints, with optic neuritis and tubercles in the choroid.

Diagnosis.—FROM SIMPLE MENINGITIS.—In the tubercular form we must be guided by the evidence of tubercular deposit elsewhere (enlarged glands, joint disease, and tubercular lesions in the lungs and choroid). The affection is also most marked at the base of the brain, causing irritation and paralysis of the cranial nerves ; whilst in simple meningitis the inflammation is mostly confined to the vertex. In the latter condition also the initial symptoms, such as fever and vomiting, are more intense, and coma supervenes earlier. In other words, it is a more acute disease. The evidence of disease of the middle ear, or of any blow on the skull, would also confirm the diagnosis. A marked tubercular family history is of much diagnostic value.

FROM ENTERIC FEVER.—The diagnosis is often difficult, especially in the early stages. Persistent headache and optic neuritis do not occur, except rarely, in typhoid fever. Also, after the first week the characteristic stools, and rash, and distended abdomen of typhoid should be sufficient to guide us.

Prognosis.—The disease is invariably fatal so far as we know. It is possible that some few cases recover, just as others recover from tubercular affections of air-passages and other parts. But the rule is that tubercular meningitis is only a local accentuation of general tuberculosis, and is not

therefore an isolated lesion. Death usually occurs within three weeks; but the patient may linger on to five or six weeks.

Treatment is very unsatisfactory. We can only relieve symptoms. Put the patient in a cool, dark room, apply ice or evaporating lotions to the shaved scalp. Purgatives, such as calomel (gr. ij.), may help to relieve the cerebral inflammation; so may blisters applied to the nape of the neck, or behind the ears. Iodide of potassium is generally prescribed with a view to modify the effects of the inflammatory process; the results are, however, disappointing. Recently the base of the skull has been trephined and the tubercular products washed out.

Bromide of ammonium may be given to relieve pain and afford some sleep.

PACHYMENINGITIS

Definition.—Inflammation of the cerebral dura mater.

Causation.—*Age.*—Most common in advanced life; comparatively rare under thirty years. *Sex.*—Males predominate. *Injuries and Diseases of Cranial Bones.*—Under this head we must include blows inflicted on the skull, fractures of skull bones, syphilitic aeries, and inflammatory extensions from the petrous or mastoid bones in ear disease. *Cerebral Congestion.*—From whatever cause, whether it be from sunstroke, alcoholism, or the like. It is probable that in all such cases a previous degeneration of the vessels had existed.

Pathology.—Two varieties of pachymeningitis are recognised: v. *P. externa* and *P. interna hæmorrhagica*. In the former instance the inflammation and its effects are limited to the interval between the skull bones and the dura mater, and is usually set up by some traumatism, by which blood is effused, and separates the membrane from the bone; or it is secondary to extension of disease from the petrous or the mastoid portions of the temporal bone, as in suppurative disorders of the middle ear.

In *P. interna hæmorrhagica* the pathology is not so clear. It has been supposed that inflammation of the dura mater

having occurred, hæmorrhage takes place subsequently in the newly-formed inflammatory tissue. Another view is that hæmorrhage is the primary condition, the clot becoming organised with fibrous tissue, into which secondary hæmorrhages infiltrate. This diversity of opinion is apparently due to the appearance of the blood tumour. It varies considerably in size, its external surface is moulded against the bones of the skull, whilst its internal surface is more convex, somewhat lobulated and limited by a capsule of fine areolar tissue containing large capillaries. Within this capsule, and also infiltrating the capsule itself, are seen layers of fibrinous clot containing blood cells and pigment, the lamination strongly suggesting the recurrence and coagulation of successive hæmorrhages.

The convolutions of the brain in the immediate neighbourhood are occasionally flattened, or pushed away from their normal relations.

Symptoms.—Headache is the most constant symptom. Its locality varies according to the seat of the lesion ; but it is generally severe owing to the free distribution of the sensory fibres of the fifth nerve to the dura mater. Other symptoms to be expected are twitchings of muscles generally, with impaired movement, and perhaps convulsions or temporary insensibility. As the hæmorrhage or the inflammatory products increase in bulk, hemiplegia gradually supervenes, and also coma, the case then bearing a strong resemblance to an attack of apoplexy. The symptoms are liable to remissions and exacerbations according as the mechanical pressure exerted by the lesion lessens by absorption, or increases by fresh mischief.

Diagnosis.—FROM APOPLEXY.—Pachymeningitis is distinguished by its previous history of causal injury, by the gradual onset of symptoms, by their remission, and by the attendant hemiplegia and coma being less profound than in apoplexy.

Treatment should be carried out according to the same rules as apply to apoplexy.

ENCEPHALITIS (CEREBRITIS ; ABSCESS)

Definition.—Inflammation of the brain substance.

Causation.—It may arise as a primary disease, though rarely ; it may be secondary to meningeal inflammation, or to caries of cranial bones. Occasionally it occurs as a manifestation of pyæmic infection ; or it may result from chronic alcoholism. But, from whatever cause, diffuse encephalitis is extremely rare. The local forms of encephalitis are usually caused by embolism or thrombosis, or by local injuries and wounds.

Pathology.—Whatever the causation may be, it must be remembered that the post-mortem appearances differ considerably in a series of cases. In the diffuse form of encephalitis there may be few or no signs to be observed after death, at least with the naked eye ; and we must therefore infer either that in such cases death has occurred before any gross pathological changes could have become manifest, or that the brain is liable to an inflammation of a lower type than that which leads to suppuration. It must not be supposed that suppuration does not occur. It does ; but the abscess is usually a small one, supervening on acute local inflammation, generally in some area which is rich in blood supply.

Local encephalitis mostly results from embolism, from thrombosis, from hæmorrhage, or from tumour. In *Embolism*, the supply of blood being cut off, and the collateral circulation poor, the cerebral substance beyond appears to perish, to undergo a kind of necrosis from want of nutriment. The brain tissue becomes pale, and soft or creamy ; it easily breaks down under a stream of water, by which an irregular-shaped cavity is formed, the walls of which only vary in consistency from the softened area, the colour of the two being much about the same. Microscopically we may, perhaps, detect some migrant leucocytes ; but the principal characteristics are roundish granule cells, and oily globules which have resulted from destruction of the myeline.

In *Thrombosis* much the same process occurs ; but, on account of the greater extent of passive hyperæmia, the thrombotic area is red or some darker colour.

In *Hæmorrhage* and *Tumour* there is also an inflammatory zone surrounding the lesion. This area may appear soft and white, as in embolism ; but its colour varies according to the amount of blood which is extravasated ; in some cases being of a pale yellow tint, in others a rusty red colour due to blood pigment.

In all cases which go on to suppuration, the softening commences in the centre of the lesion, the histological elements become invaded with leucocytes, and eventually pus forms.

Symptoms.—The symptoms in the main resemble those of meningitis, with some slight modifications. The patient complains of *headache*, which is often local in character, and occurs in paroxysms ; but is not so severe as in meningitis. He also has *pyrexia*. He is restless and irritable ; the face is flushed ; the pulse feeble and irregular ; the skin hot and dry ; the tongue furred ; and there is usually constipation. Here again, however, the febrile symptoms are less pronounced than in meningitis. The patient is also liable to subjective symptoms, such as roarings and buzzings in the ears, double vision, flashes of light, and the like ; these apparently being dependent on the locality of the inflammation.

As the disease advances, there are tonic spasms affecting the muscles of the eyes or limbs, and the pupils lose their reflex action to light. The patient is not, as a rule, delirious, but rather tends to imbecility, with impaired speech, loss of memory, incoherence, and temporary insensibility. Subsequently twitchings of muscles and cutaneous insensibility supervene, and finally complete paralysis of motion and sensation with unconsciousness.

The above is an account of the symptoms which are generally observed ; but they will obviously vary according as the inflammation is general or local only.

Diagnosis.—FROM APOPLEXY.—In this disease the suddenness of the attack, with insensibility, stertor, a full bounding pulse, and signs of hemiplegia, would usually enable us to distinguish it from encephalitis.

FROM MENINGITIS.—The diagnosis is one of great difficulty, the two conditions being usually associated. In encephalitis

the fever and headache are less, irritation of cranial nerves not so pronounced a feature, whilst impairment of speech and other mental functions are prominent.

Prognosis.—Unfavourable, except when the encephalitis is the result of injury, or when it results in abscess which is near the surface and amenable to surgical treatment.

Treatment.—Little can be done beyond applying cold to the head, with blisters to the nape of the neck and behind the ears. Bromides may be prescribed to relieve the headache. Chloral hydrate is the best sedative. Depletory measures should be avoided. The question of surgical operation to evacuate any superficial abscess may have to be considered. The other general rules of treatment are the same as in meningitis.

EMBOLISM AND THROMBOSIS OF CEREBRAL ARTERIES

Definition.—Cerebral embolism is an internal obstruction to an artery or capillary of the brain, caused by some migrant foreign body which acts as a plug.

Cerebral thrombosis is an internal obstruction originating *in loco*. This may occur in the cerebral sinuses as well as in the arteries.

Causation.—Embolism may occur at all ages, though most frequently in *youth*. In most instances it is due to the detachment of a vegetation from one of the valves, or from the interior of the cavities, of the left side of the heart. A portion of a thrombus even may become separated and act as an embolus.

Thrombosis is usually a condition found in *old age*; but it is not uncommon in middle life, when due to *syphilis*; and it may occur in children as a sequel to some *specific fever*.

In a general way we may regard thrombosis as taking its origin either in (a) some altered condition of the blood itself, which predisposes to intravascular clotting, or (b) some inflammatory or degenerative changes in the walls of the vessels themselves.

Nevertheless, an embolon itself may, by its obstruction, become the centre of a fresh thrombosis on each side of it.

Pathology.—In CEREBRAL EMBOLISM the plug usually is situated at the bifurcation of an artery, neither of the branches resulting from the bifurcation being sufficiently wide to carry the migratory foreign body. The middle cerebral artery is its most frequent site, since an embolon, from say the mitral valve, finding its way into the common carotid artery is much more probable than its travelling down the subclavian to its vertebral branch; and once inside the internal carotid, the anatomical chances are greatly in favour of its travelling into its largest terminal branch. As a result of the arrest of the embolon, the brain area supplied by the plugged vessel, as a rule, perishes, the necrosed zone becoming soft or creamy in consistence, and white or yellowish in colour, according to the amount of blood which is extravasated during the process of softening. Occasionally this softened patch actually breaks down, leaving a small, irregularly walled cyst, filled with clear or turbid fluid, or with blood. Frequently the brain tissue immediately around the necrosed portion shows some secondary inflammatory changes.

In CEREBRAL THROMBOSIS coagulum being deposited, from whatever cause, on the inner wall of the vessel arrests the blood current in two ways; by its own bulk, and by the local endarteritis which it causes. Occlusion of the vessel rarely occurs at once; but successive laminae, being deposited from the blood stream, tend towards it. When complete occlusion occurs, the clotting extends forwards for some distance along the collapsed vessel, and also backwards to the giving off of the last collateral branch. The clot itself may subsequently shrink, or become organised, or undergo calcareous changes.

The local effects of thrombosis are much the same as in embolism.

Symptoms.—(1) EMBOLISM.—In a typical case there are no prodromata. Suddenly in a young person with a history of valvular disease of the heart, or of previous acute rheumatism, there is intense headache, possibly a sudden cry,

and more or less unconsciousness ; and on recovery, he is found to be hemiplegic. Subsequent rigidities occur as after apoplexy.

One or two features of the case require further comment. A valvular lesion may exist, or there may be endocardial vegetations, and yet no valvular murmur. A history of previous, it may be long previous, acute rheumatism should be always inquired about. Unconsciousness may be entirely absent, or only of slight degree. The cerebral symptoms are limited to the arrest of function of those parts of the brain only which are supplied by the plugged vessel ; and there is no centrifugal extension of damage, as in the destructive outburst of hæmorrhage. Frequently we have evidence of emboli elsewhere, especially in the spleen and kidneys, and occasionally in the retinal artery. If the left middle cerebral artery be plugged, we should find not only right side hemiplegia, but aphasia, the vascular distribution to Broca's convolution, internal capsule, and angular gyrus being arrested.

Right-sided hemiplegia as a result of embolism has been said to be more frequent than left-sided, chiefly on the anatomical grounds that an embolon entering the innominate is quite as likely to enter the right subclavian as the right common carotid ; and also that the mouth of the left carotid, on the arch of the aorta, is in the direct line of the stream impelled by the ventricle. This is probably more fanciful than real.

(2) THROMBOSIS is usually preceded by drowsiness, numbness, or affection of sensation on one side of the body, and deficient memory or other mental impairment. Coma is usually absent, or if present is mild in degree only. The onset of paralytic symptoms is gradual, as the lumen of the vessel is rarely completely obstructed at once. In large and extensive thromboses, however, paralysis may be complete and rapid. There is usually evidence of atheroma or other degenerative changes in the vessels. Lastly, there is in old-standing cases frequently a history of several recurring attacks with intervening periods of amelioration in the symptoms ; but ultimately mental failure supervenes.

Prognosis.—In EMBOLISM the immediate prognosis is

favourable, on account of the youth of the patient. Complete recovery of the use of the palsied muscles is, however, rare.

In THROMBOSIS recovery is common and often very rapid, especially in syphilitic cases.

Treatment.—The first essentials are rest and quiet. The bowels should be relieved daily without strain or effort. Give purgatives (calomel, gr. iv.) if necessary. The food should be light and unstimulating. Venesection is not usually advocated ; but we may resort to it with advantage in cases presenting a full hard pulse and signs of a plethoric or congested habit. Active anti-syphilitic treatment is necessary in thrombosis of syphilitic origin. Give mercury and iodide of potassium internally, and apply mercury by inunction so as to produce speedy results.

After active mischief has subsided, the palsied and contracted muscles may be stimulated by the faradic current. Treatment should be directed against the supervention of subsequent attacks. (See Apoplexy ; and Hemiplegia.)

THROMBOSIS OF CEREBRAL VEINS AND SINUSES

Causation.—All cases may be included in two categories, viz. : (i) those in which the primary cause is some local affection of the walls of the vessels ; and (ii) those in which some disorder of the blood is the principal factor.

LOCAL CAUSES originate chiefly in inflammatory extensions from the bones, especially of the middle ear, to the sinuses, by which a local phlebitis is set up, which ends in thrombosis and plugging of the vessel. But a similar condition may be brought about by injury to the cranial bones, also by the local pressure effects of intracranial tumours. Erysipelas of the scalp may also cause cerebral thrombosis in two ways : it may be included as a cause under the next heading, or it may cause thrombotic obstruction to those external veins of the scalp which communicate with the internal veins and sinuses. In similar manner, facial carbuncle, orbital abscess and cellulitis may also produce thrombosis. The lateral and petrosal sinuses are most frequently obstructed.

BLOOD DISORDERS, such as occur in specific fevers, may

also set up thrombosis. In our experience it occurs most frequently as a sequel of typhoid and scarlet fevers, and erysipelas, in the order named. In children it not infrequently supervenes on chronic diarrhœa, tuberculosis, and other wasting diseases. In adults it complicates tertiary syphilis and carcinoma, quite independently of any of the new formations of these diseases.

The superior longitudinal sinus is most frequently involved.

Symptoms.—There are no definite symptoms until the obstruction is well advanced. Coma comes on gradually, and increases. Spasm and twitchings of muscles of the neck and face are common, owing to cortical irritation. Subsequently signs of obstruction of the venous current appear. The veins of the scalp, forehead, eyelids, conjunctivæ, and mastoid regions become engorged and distended. Epistaxis is common, and affords relief by means of the communication between nasal veins and longitudinal sinus through the foramen cæcum. Optic neuritis, however, does not often occur, as the ophthalmic veins freely communicate with the facials.

Should the thrombosis extend from the lateral sinus to the jugular vein, it would give rise to a deep-seated tumour, which might suppurate and cause pyæmic or other forms of embolic infarction of the lungs.

With these signs there is, in addition, some febrility. The pulse is quickened, as a rule ; but it may be irregular and even slow, as in meningitis. As insensibility increases, the patient passes his urine and evacuations in bed ; and death occurs either in profound coma, or from the effects of pyæmic infection of the lungs.

Treatment.—Of recent years the treatment of thrombosis of the cerebral sinuses has been, in the main, surgical. The mastoid cells, when they are the seat of the original disease, may be trephined and their contents evacuated. Similar operations have been successfully performed in relation with the lateral sinuses. Strict antisepsis is, of course, an imperative necessity.

In the early stage hot fomentations to the scalp often

afford relief. Leeches may also be applied to the forehead, or behind the ears, so as to relieve venous engorgement.

Medicines in our experience have not afforded much success. Most practitioners give large doses of iodide of potassium with a view to promote absorption of the clot. It may with advantage be combined with free doses of carbonate of ammonia. We also agree with Bristowe that, on account of the intense pain which often attends this disorder, opium is not contra-indicated, and usually affords relief.

APOPLEXY (CEREBRAL HÆMORRHAGE)

Definition.—A sudden unconsciousness, with loss of motor power and of sensation.

The term is usually confined to this condition when supervening on cerebral hæmorrhage ; and for similar pathological reasons we speak of apoplexy of lung, spleen, placenta, &c.

Apoplexy may be due to causes other than hæmorrhage, such as concussion, uræmic and other poisons. The following description, however, is limited to apoplexy due to cerebral hæmorrhage.

Causation.—**PREDISPOSING.**—*Age.*—Most frequent between forty and sixty years of age, at a period when, although tissue changes are beginning to show themselves, severe exertions and bodily exposures also act as exciting causes. After the sixth decade, although vessels are still more prone to rupture, the surroundings of a man's life are usually of a more reposeful character. *Sex.*—Men much more frequently than women. *Diseases of the Blood.*—Notably purpura ; also small-pox and other fevers. *Diseases of the Vessels*, such as fatty, calcareous and other degeneration, aneurysm, vascular changes produced by Bright's disease and syphilis. *Traumatic Injuries*, under which heading would be mentioned fractures of skull, and congestions and lacerations of the brain substance. Concussion, although not necessarily attended by the effusion of blood, may be mentioned here. *Changes in the Cerebral Matter*, including inflammation and softening, by which the natural support in the vessels is lessened, hence allowing of bulgings and dilatations. The effects of embolism

and thrombosis may also be included in this category. *Alterations in Vascular Tension*, such as any sudden increase of the force of the blood, or any impediment to the return of blood to the heart. This may be brought about by alcohol, mental excitement, straining during parturition, or at stool.

Pathology.—Cerebral hæmorrhage is a wide term so far as pathology is concerned. It includes all those cases which may be caused by (1) *injury*, the vessels being quite healthy ; and another large group, in which the direct cause is (2) *disease* of the arteries. On this division hangs another classification which is mainly anatomical, viz. the *site of the hæmorrhage*.

(1) Blows on the skull, or fractures of its bones, may cause cerebral hæmorrhage. The bleeding point, however, is usually found in one of the meningeal vessels, unless indeed the injury is so extensive as to cause rupture of a cerebral vessel. In these cases the hæmorrhage, often extensive, may be confined between the bone and dura mater, or it may infiltrate the arachnoid cavity or subarachnoid space. The rule is, however, that hæmorrhages due to injury are superficial, and that their effects are rather those of cortical pressure than of destruction of brain substance.

(2) Disease of the vessels is always most marked at the base, in the circle of Willis, or in the large arteries which contribute to form it. Atheroma, calcareous degeneration, and syphilitic arteritis are the most frequent causes ; and as these pathological changes are not limited to the larger trunks, but extend to the smaller branches, a vessel may rupture on the cortex, in the centrum ovale and in the basal ganglia. In the two last situations there is a tendency, if the hæmorrhage be large, for it to extravasate into the lateral and communicating ventricles. One artery especially appears prone to rupture, viz. the lenticulo-striate branch ('artery of hæmorrhage') of the middle cerebral artery, which passes between the lenticular nucleus and the external capsule. It is obvious that the amount of hæmorrhage will vary according to the size of the vessel and the extent of its rupture. Thus we may have merely a perivascular extravasation, the vascular sheath being filled with blood, and causing only slight symptoms ;

or there may be a slight extravasation, sufficient to cause marked apoplectic symptoms, but not enough to cause any marked destruction of cerebral tissue. This form heals by cicatrisation, the scar becoming eventually slightly puckered and discoloured. Finally there is the massive hæmorrhage, which, starting usually from some vessel in the corpus striatum or optic thalamus (35 per cent. of cases), ploughs its way into the centrum ovale or into a lateral ventricle, and, causing the gravest symptoms, usually ends in death. It would appear from post-mortem room experience, that a heavy percentage of these large hæmorrhages is due to the rupture of small aneurysms in the branches of the cerebral arteries, and that they are not sufficiently searched for, as they are not suspected.

If the patient die within a short time, as is usual, the situation of the hæmorrhage will be found by making horizontal sections of the cerebrum down to the ventricles. This is on the supposition that the lesion has occurred at the most frequent site. It must, of course, be remembered that rupture of a vessel may occur in the cerebellum or in the pons or medulla, but more rarely in these situations than in the cerebrum.

At the lesion the brain substance is ploughed up into an irregularly-shaped cavity, varying in size according to the resistance of the tissues and the channel of outlet of the blood. It is filled with a dark jelly-like clot, which fits the lacerated irregularities of the cavity walls, and perhaps pushes the neighbouring velum, or corpus callosum, or convolutions on one side.

If the patient survive for any length of time, and die from, say, a fresh hæmorrhage or some other cause, the old hæmorrhagic cavity may still be discovered, but somewhat contracted in size, its walls smoother and stained with hæmatoidin, whilst the interior is filled with the shrivelled remains of a clot, or by some turbid milky serum, with a delicate areolar tissue intermixed. Small cysts are not infrequently found.

The subsequent effects of a hæmorrhage from which the patient recovers would be seen in descending sclerotic changes in the cord (see Lateral Sclerosis). The brain itself might be

subjected to œdema or to secondary inflammatory changes of the part around the hæmorrhage, which is possibly discoloured, its convolutions displaced, and its sulci unfolded and obliterated. It will thus be seen that the effects of hæmorrhage in the interior of the brain are, destruction of brain substance, and the secondary effects of pressure.

Symptoms.—The onset may be sudden, with no premonitory symptoms whatever. It will then be generally found that the vascular system is profoundly diseased, and that the consequent hæmorrhage is a large one. Ordinarily, however, there is a prodromal stage, although it may be overlooked. This stage is marked by drowsiness, vertigo, headache, mental confusion, irritability of temper or loss of memory. Or the patient may complain of numbness, or of slight loss of power in one arm or leg. Occasionally there is some thickness of speech, or difficulty in deglutition, or a complaint of biting one side of the tongue during mastication.

One or more only of the above symptoms may be present, and may precede the true attack by a longer or a shorter period.

Nor are they, singly or collectively, necessarily diagnostic of oncoming apoplexy ; a like chain of events takes place in cerebral tumours.

The grave attack itself is generally ushered in with a sudden syncope, with perhaps vomiting, and a cold clammy skin. To this succeeds a convulsive attack, resembling in most of its features an epileptic seizure. The patient utters a cry or groan and falls to the ground completely unconscious, with loss of sensation and of motor power. All these symptoms are due to pressure suddenly exerted within the cranium ; and consequently muscular spasm, or loss of consciousness, will depend, so far as their extent and degree are concerned, on the seat of the hæmorrhage. The patient, however, lies in a state of coma ; he cannot, as a rule, be roused even by deep stimulation ; his breathing is loud and stertorous ; his face is turgid, and expressionless from one-sided paralysis of its muscles ; the eyelids are closed ; the pupils fixed either in dilatation or in contraction ; deglutition is impaired ; the limbs are rigid and motionless ; the reflexes are absent ; the skin is

bathed in cold perspiration ; his pulse is full and throbbing ; the urine is retained till it dribbles away ; he passes his fæces involuntarily ; and his temperature has fallen probably one or two degrees below normal. From this stage he may gradually recover. Consciousness slowly returns ; his pulse becomes softer ; his temperature is restored to the normal level, or even exceeds it ; he regains some control over the sphincters ; his breathing becomes less noisy and laboured ; and he shows some recognition of his friends and surroundings. He recovers from the attack, however, only to find himself helpless or paralysed in one lateral half of the body, and, it may be, aphasic. The above condition is vulgarly known as an 'apoplectic stroke.'

On the other hand, if the case is about to terminate fatally, the symptoms increase in severity. The pulse becomes quicker but feeble ; respiration is shallow and slower ; mucous secretions accumulate in the bronchi and impede respiration ; the temperature rises to 103° or 104° ; muscular twitchings are frequent ; the pupils dilate ; coma is still a marked symptom ; and finally the patient dies, apparently from collapse.

The above description, however, requires some amplification in its details. There is not necessarily any order in which the symptoms supervene. Hemiplegia may even precede insensibility. A cry or groan, as the patient falls, is usually to be heard. Insensibility, when it supervenes, is immediate and usually profound, the patient rarely being roused by any stimulus. This is especially so in large hæmorrhages on the surface of the brain, or in the pons. But every degree of stupefaction is met with. Stertorous breathing and puffing of the cheeks are caused by paralysis of the palate and cheek muscles. They both indicate deep stupor, and are usually symptoms of grave omen. Occasionally the respiration assumes the Cheyne-Stokes type.

The pulse, though usually full, slow and bounding, may be irregular and intermittent. It rises to 120, or more, towards the end in fatal cases.

The pupil, though fixed, does not respond to light, except in favourable cases.

Deglutition of fluids is not impossible. But it is always impaired, and there is a danger of food passing down the air-passages. The power of mastication, however, is always lost.

The muscular convulsions may vary in different cases ; in some there are tonic, in others clonic, spasms ; occasionally the limbs are flaccid and limp, but rigidity is the most usual and especially affects the arms (early rigidity). The paralysis is the direct mechanical result of the hæmorrhage.

The urine is retained ; and although after a time it dribbles away from overdistension of the bladder, this organ may, from neglect, become enormously distended or even ruptured. The fæces are, however, passed involuntarily.

In all cases the temperature is lowered about 2° ; it is never restored to its normal level in fatal cases. A few days before death occurs it may rise to 103° or 104° . But a return of the temperature to normal, or slightly above, is a favourable sign.

Diagnosis.—Cerebral hæmorrhage may be confounded with cerebral embolism, with cerebral tumour, and with the various comatose conditions due to uræmia, glycosuria, epilepsy, compression, or to opium and alcohol poisoning.

EMBOLISM would be suspected in a young subject with a previous history of acute rheumatism, and suffering from chronic endocarditis, or perhaps aortic disease. The attack is sudden, without any premonitory symptom ; but consciousness is not necessarily lost.

In **CEREBRAL TUMOUR** the onset is gradual ; vomiting, persistent headaches, and various irregular paralyses precede coma. The diagnosis would also be assisted by an absence of any signs of disease in the kidneys and cardio-vascular system.

COMA.—When dealing with a case of coma without any previous history, the diagnosis is by no means easy. The following points, however, may help.

In **URÆMIA** the convulsions are frequent and general ; they precede any definite coma, and may consequently simulate any attack of epilepsy ; the temperature is subnormal and does not become febrile. Confirmatory evidence would be gained by finding albuminuria, œdema of the eyelids and ankles, and the cardio-vascular changes of Bright's disease.

DIABETIC COMA may supervene suddenly ; but the odour of the breath, the bodily emaciation, and the presence of glycosuria would be diagnostic.

In OPIUM POISONING, coma only supervenes on an insensibility from which the patient can at first be roused ; that is to say, the coma is not suddenly complete. Stertor is not so marked a symptom, but the pulse and respirations are extremely slow. The pupils are more contracted than in apoplexy ; they are often no larger than a pin-point. The skin is bathed with cold sweat, but the other secretions are arrested.

In ALCOHOLIC COMA there is usually a previous history of boisterous excitement or struggling. The coma, except in rare instances when large quantities of raw spirits have been taken, is not so profound as in apoplexy, or in opium poisoning. The pupils are contracted if the patient is left undisturbed ; they dilate when the patient is shaken or roused, but return slowly to the contracted state afterwards (MacEwen). The odour of alcohol is not conclusive, and even may mislead, as spirits may have been given to the patient, no matter what was the nature of the 'fit.' A good rule is to invariably regard coma, even if there is a strong supposition of its being alcoholic, as a most grave condition.

In EPILEPTIC COMA the tongue will probably be bitten, the evacuations passed in the clothes, and the patient will gradually recover. Often a history of a previous fit may be obtained.

In COMPRESSION from bone injury, the coma is instantaneous ; if from gradual effusion of blood due to injury, the coma only comes on after a few hours ; if from the pressure effects of pus, coma supervenes only after some days or even weeks.

Coma attended by hemiplegia is in the majority of cases due to hæmorrhage.

In all cases in which the diagnosis is doubtful, it is essential that the condition of the urine be carefully investigated, and that all vomited matter be examined.

As to the diagnosis of the *situation* of the hæmorrhage, the following table is a fair guide ; but the symptoms are by no means constant.

Cortex Severe convulsions, followed by profound coma.
External capsule .	. Fainting, partial recovery, followed by coma.
Internal capsule .	. { Coma, partial recovery, with more or less complete hemiplegia of opposite side; and possibly aphasia.
Corpus striatum .	. { Motor paralysis complete, but unconsciousness not so marked.
Crus cerebri . .	. { Paralysis of the third and fourth nerves on the same side as the lesion.
Pons or medulla .	. { Severe convulsions as in epilepsy, except that they are bilateral; the pupils extremely contracted; probable paralysis of the fifth, sixth, and seventh cranial nerves on the same side as the lesion.
Cerebellum Staggering or gyratory vertigo, severe vomiting.

Prognosis.—Not only is the attack itself a grave disorder, but the bodily condition which predisposes to an apoplectic seizure is always one of anxiety.

Death rarely occurs within a few hours from the onset of the attack; usually the fatal termination occurs about the second or third day; the survival over the eighth day of a case which eventually proves fatal is rare.

Many cases do recover perfectly, especially from an initial seizure; but all subsequent attacks render the chances of recovery more remote. With the public it is a common idea, and in the main a correct one, that a patient never survives a third attack. The probability of survival will be greatly influenced by the previous history of the patient, and the amount and the locality of the hæmorrhage. Hæmorrhage into the ventricles or into the pons is almost always fatal. Amongst grave symptoms may be mentioned, absolute inability to swallow even liquids, severe muscular convulsions, an irregular pulse, marked puffing of cheeks, ‘Cheyne-Stokes’ type of respiration, mucous râles in the larger bronchial tubes, and after a few days a rise of temperature to 103° or 104°. Prolonged insensibility, say for twenty-four hours, or a return of the insensibility, indicating a fresh hæmorrhage, is invariably followed by death.

But there is a further point in prognosis, and that is, suppose the patient recovers from the present attack, will he be

paralysed, or will his mental condition be unimpaired? These are questions of great anxiety to his friends and relatives. As a rule, we may divide those cases which survive the seizure into three groups, viz : (i) some are restored mentally, but with partial or complete paralysis of one leg, arm, and side of the face ; (ii) some recover from the immediate effects of the attack, but remain hemiplegic, and become mentally defective ; (iii) others recover entirely. This may occur even though the fit was attended by marked stupor for some time. It is rare, however, as some function is usually impaired.

Complications and Sequelæ.—It is obvious that the effects produced by cerebral hæmorrhage will vary considerably in different individuals, according to the situation of the lesion and the extent of its damage. The most usual result is hemiplegia of that side of the body opposite to the lesion. In some cases it is attended by hemianæsthesia. In others the attack may leave no signs of paralysis whatever. Hemiplegia must follow, however, if the motor tract of the internal capsule be injured ; and similarly we find hemianæsthesia when the posterior portion of the internal capsule is involved.

Bedsore may form on the hip, sacrum and shoulder of the paralysed side (a grave sign).

The hæmorrhage may act as an irritating foreign body, and, as such, produces a surrounding zone of cerebral inflammation, with a return of symptoms which are entirely due to this local cerebritis. This would be indicated by febrility, drowsiness, a return of coma and paralysis, and eventually death.

Or, whilst the effects of the primary hæmorrhage are subsiding, a secondary rupture may occur from the original or from some other vessel. Recovery is rare in such instances.

Occasionally convalescence is complicated by pneumonia, enteritis, or other inflammatory affections of viscera.

Hæmorrhage involving the third left frontal convolution and internal capsule, besides causing right hemiplegia, is attended by difficulty of speech, with indistinct utterance or complete aphasia.

From two to three weeks after the attack, the effects of cerebral irritation may be seen in rigidity and wasting of

muscles on the palsied side (late rigidity), followed by spasms and contractures, or by athetosis and other movements.

Lastly, mental defects may supervene. The patient is apt to become emotional, his memory fails, and he becomes stupid or childish in his manners, or dirty and careless in his habits.

Treatment.—(A) DURING THE SEIZURE.—In a hospital the patient is simply placed on a bed in a cool ward, all tight clothing removed, and all noise and other disturbing elements excluded; his head may be shaved, and an ice-bag applied. A purgative of croton oil (ʒij.) or of calomel (gr. v.) may also be given. As Suckling rightly says, ‘the treatment of apoplexy is one of non-intervention.’

Frequently, however, more active procedures are demanded. The patient’s feet may be placed in hot water; sinapisms may be placed on his calves. If there are signs of high arterial tension the patient may be freely bled. We may also assist by dry cupping, or by free purgation. He should be rolled on to his paralysed side, if there be any signs of respiratory trouble. But all further treatment should be directed towards keeping the patient at perfect rest. Only a spare milk diet, if the patient can swallow, should be allowed.

(B) AFTER THE SEIZURE.—Rest is still important. The patient’s mind should, as far as possible, be relieved from care and excitement. The bowels should act freely every day. All symptoms of pain and headache should be treated by bromides. Opium and stimulants should, of course, be avoided.

The diet should be simple and nutritious, large meals being forbidden. The paralysed muscles require treatment only after all acute and irritative signs have passed away.

Faradisation, with alternations of shampooings with stimulating liniments, should be applied to the palsied limbs, with a view to increase the nutrition of the muscles.

CEREBRAL TUMOURS

Causation.—The brain and meninges may be the seats of new growth and parasitic invasion in common with other organs and parts of the body. And although certain neo-

plasms, such as glioma and psammoma, appear to have a special tendency to flourish in the central nervous system, it is not exempt from any of the other general tumours. The most frequent are, tubercle, syphilis, malignant new growths (glioma, sarcoma, cancer), aneurysm (miliary), and the cystic stage of the various tapeworms.

It must be remembered, from its important bearing on school life, that a blow or an injury to the head may render latent tubercle active, or even, according to Suckling, 'cause the development of a glioma in the brain.'

Pathology.—TUBERCULAR GROWTHS are most common. They vary in size, occasionally growing to the bulk of a walnut, or larger; they also may be multiple, but are often single. They do not tend to undergo caseous degeneration and liquefaction so frequently as elsewhere. They occur especially in children, and in the cerebral or cerebellar cortex. Miliary tuberculosis is generally associated with meningitis.

SYPHILITIC GROWTHS are generally multiple. They spring most commonly from the dura mater, but are found in association with the pia mater, and also embedded in the brain substance. They are always tertiary manifestations. The effects which they cause are mainly those of pressure; but they also tend to involve the arteries at the base causing obliterative arteritis and thrombosis. The brain tissue in their neighbourhood is thus impoverished, whilst the syphilitic growth itself is surrounded by an area of inflammation.

Glioma is a new growth closely connected with the sarcomata. It occurs most frequently in children. It is usually a largish, single growth, springing from the retina or from the brain substance. The connective tissue of which it is mainly composed closely resembles neuroglia, and as the growth is extremely vascular it is usually of soft consistence and prone to hæmorrhage. Other malignant or semi-malignant tumours occur, but are rare.

Aneurysms may develop from the basilar, from the internal carotid, both at the base of the brain and during its passage through the temporal bone, and also from the other cerebral arteries. Indeed, no artery within the cranium is exempt from

aneurysm. Their size varies from a hemp seed to a large bean. They appear to follow the general rule of aneurysmal dilatations, viz. that the smaller saccular varieties are more prone to rupture than the larger fusiform ones. Not infrequently an aneurysm is found immediately behind an obstructing embolism. As would be expected, they are more prevalent in late adult life, when tissue decay is commencing.

Hydatids and other cystic parasites may be single or multiple, and develop in the brain substance or its membranes; no part being exempt. Their effects are mainly those of pressure, their walls being extremely tense and resistant. They are comparatively rare pathological curiosities.

Symptoms.—**HEADACHE** is the earliest and most prominent symptom. It may be paroxysmal or constant; and at different times is shooting, or bursting. Indeed, all the different qualities of headache may be present. Not infrequently it is the only symptom which is complained of for some long period. Its situation is not necessarily a guide to the locality of the tumour.

VOMITING is also invariably present at one or other period, but especially in the early stages. It is frequently very severe, and often without premonitory nausea, and has no relation to food.

VERTIGO is rarely absent. In cerebellar tumour it is usually gyratory.

IMPAIRMENT OF VISION, occasionally amounting to complete blindness. Double optic neuritis is almost invariably present.

PRESSURE SIGNS.—These will vary according to the situation and extent of the tumour, and the adaptability of structures which are involved by it. Convulsions supervene sooner or later, and they may be general, as in epilepsy, when the tumours are multiple or diffuse, or they may be local but persistent, involving one arm or a certain set of muscles, as in Jacksonian epilepsy. Towards the end of the case, the convulsions often cease, to be succeeded by the fatal coma. Another effect of pressure is seen in oedema, which may involve the vessels of the eye and nose, owing to pressure on the cere-

bral sinuses. Protrusion or complete disorganisation of the eyeballs may also result.

Lastly, the effects of pressure may be seen by its giving rise to hemiplegia, or to various forms of monoplegia. Frequently these symptoms resemble those found in the initial stage of apoplexy. That is to say, the patient may complain of numbness in one arm and leg, followed by more or less complete hemiplegia and hemianæsthesia. The arm is affected oftener and more deeply than the leg, following the ordinary rule in hemiplegia. Or there may be some hesitation and thickness in speech, or difficulty in deglutition.

Further, and this is very suspicious of tumour, these hemiplegic symptoms may come abruptly, and almost as suddenly disappear. This is probably due to some forms of new growth which increase first in one direction and then in another.

Pressure on a cranial motor nerve is revealed by palsy of those muscles which it supplies. It is comparatively rare for one nerve alone to be injured, but it is not uncommon for one or more cranial nerves on the same side to be involved. But this, of course, depends on the situation of the tumour, since if it be limited to one side of the base of the brain, only those nerves of that side would be affected, whilst the opposite side of the body is hemiplegic ; whereas a tumour in the base of the brain in the interpeduncular space would impinge on the trunks of nerves of both sides, from the second to the fourth at least (see fig. 27).

Similarly pressure on a cranial sensory nerve will be shown by disturbance or abeyance of its special functions, such as double vision, total or partial blindness, flashes of light ; or by deafness or drummings and other distressing noises in the ear ; or by hyperæsthesia or total anæsthesia in those parts which are supplied by the motor part of the fifth nerve. Again, injury to the fifth nerve in front of the Gasserian ganglion may lead to sloughing and disorganisation of the eyeball.

It is unnecessary to go into further details as to the pressure effects upon nerves, beyond saying that it is rare for one nerve alone to be implicated ; that they become involved often

in an irregular and confusing sequence, which is in itself a suspicious diagnostic sign.

MENTAL DISTURBANCES.—Often in the early stages there are no mental symptoms whatever. One frequently sees children suffering from cerebral tumour who are completely blind, and who suffer from various paralyses, and yet who are contented, cheerful, and indeed bright. But sooner or later perception and sensation become blunted ; the memory fails ; speech becomes thick, or fails entirely ; and then follow convulsions with unconsciousness. The patient may partially recover at this stage, the coma passes away, convulsions are less frequent, and the patient appears about to be restored to health. But eventually, as the tumour increases, or as its effects become more diffused, the patient's mind fails ; he may become stupid and demented, or maniacal, until death occurs.

Death is often preceded by hydrocephalus or the symptoms of cerebritis. The course of the disease is always slow to the fatal ending, although sometimes accelerated by bedsores and convulsions.

The various cerebral reflexes are lost if the lesion is situated above their nuclei. Thus, in a lesion involving the parts above the corpora quadrigemina, the pupil is fixed in dilatation ; but the iris reflex is present when the growth is in relation with the optic tract.

The above is merely an outline of the chief symptoms ; many of them may be absent ; and, on the other hand, we saw under Dr. Bristowe, in St. Thomas's Hospital, a patient with all the typical signs of cerebral tumour, but none was found at the autopsy. The most reliable symptoms, beyond headache, vomiting, vertigo, and optic neuritis, are the gradual onset of symptoms of paralysis of certain cranial nerves, step by step, together with, ultimately, spasm, rigidity and paralysis of the muscles and the regions which they supply.

The temperature and pulse are not affected by the new growth, unless as a consequence of the surrounding inflammatory zone which they set up.

Diagnosis.—FROM APOPLEXY.—In this condition we are

guided mainly by the advanced age of the patient, and by the symptoms being sudden in their onset and complete in their character. Coma and paralysis appear at the same time. There is no extension of palsy from one set of muscles to another. Confirmatory evidence would be afforded by any existence of kidney or of cardio-vascular disease.

FROM ABSCESS OF BRAIN.—There would have been a history of rigors, and irregular temperature; pain is not so pronounced a symptom; often there are no marked signs until meningitis supervenes. Examine the ears to ascertain existence of otorrhœa; often there is a history of bone disease or of suppuration (pyæmia).

The diagnosis of the nature of the tumour is only possible when the cerebral symptoms agree with, or are suggested by, the symptoms of disease existing in other organs.

Thus, in a young subject with phthisis, it is fair to presume, in default of any contradictory evidence, that the cerebral tumour is tubercular. Or in a middle-aged man with, say, a gummatous testicle, the cerebral growth is probably of a similar nature. Again, in an elderly woman who has been the subject of mammary cancer, the suspicion is strongly in favour of a cerebral tumour being carcinomatous. Similarly the existence of an hydatid cyst, say in the peritoneum, suggests that any symptoms of cerebral tumour may be caused by the same parasite.

After the tumour, whatever its nature, has attained a certain size, the symptoms which it produces are similar and common to all, and it tends to destroy life by the effects of direct cerebral pressure, such as inflammatory softening, venous engorgement, and irritative lesions.

Treatment.—As a rule unsatisfactory. So far as the mechanical effects of the tumour are concerned, all that we can do is to relieve the symptoms produced. Pain may be relieved by bromides, or by opium carefully watched. The patient also often finds relief by being placed in a cool, dark room, with an ice-bag applied to the scalp. Check vomiting by effervescent medicines combined with dilute hydrocyanic acid. The bowels should be relieved daily and without effort.

A suitable bed or cushions should be ordered, with a view to arrest the formation of bedsores.

Iodide of potassium and mercury in full doses are indicated in syphilitic growths and also when symptoms lead us to the diagnosis of aneurysm. Under this persistent treatment the prognosis of syphilitic tumour is not without hope.

The removal of tumours from the membranes of the vertex, and from the more accessible portions of the brain, has recently been performed with marked success. No patient should be allowed to perish without operation, provided surgery holds out reasonable grounds for hope of relief.

HYDROCEPHALUS

Definition.—Dropsy of the ventricles of the brain. This term, however, is also applied to those conditions in which there is an excess of arachnoid fluid on the surface of the brain, generally associated with arrest of development; and also to certain malformations in which portions of brain or of its membranes form a cyst which protrudes through some unclosed aperture or suture in the cranium (hydrencephalocoele, meningocele). There is also an excess of the subarachnoid fluid in certain forms of meningeal congestion and inflammation.

The intraventricular dropsy to which the following description applies is, however, essentially a congenital condition, or it supervenes shortly after birth.

Causation.—No absolute cause is known. By some it is regarded as the sequel of acute intra-uterine meningitis. It has also been attributed to œdema, caused by pressure on the straight sinus, or on the vena Galeni; also to the effects of tubercle. In the majority of cases, however, no attributable cause can be assigned.

Pathology.—The fluid occupies the cerebral ventricles. The quantity may vary from a few ounces to six pints, or more. The fluid contains albumin and chlorides, and has a specific gravity from 1006 to 1014. The accumulation is an intra-uterine pathological change or, at latest, it commences between birth and the primary dentition. The effects of

the fluid are seen in the mechanical distension of the ventricles, by which their dividing walls and foramina of communication may be obliterated, the basic ganglia flattened and compressed, the sulci unfolded, and the convolutions flattened out. In short, the greater portion of the brain above the lateral ventricles may be converted into one large cyst, with thin walls of compressed grey matter ; a condition comparable to that seen in hydronephrosis. It is not always, however, that the accumulation is symmetrical. Adhesions may close the foramen of Monro and the aqueduct of Sylvius, and thus confine the fluid to one lateral ventricle, with consequent pressure effects on one hemisphere of the brain only.

Symptoms.—The early stage of the disease is not always recognised, unless the head be unduly enlarged at birth. Usually the symptoms show themselves some few months after birth, and commence with convulsions or constant head pains. The most striking symptom is enlargement of the cavity of the cranium. The enlargement occurs at the expense of all parts of the skull except its base, but especially in the direction of its upper and lateral walls. The reason of this is a purely mechanical one, as the base and anterior walls are more rigid, and offer more resistance than other parts. But even the roofs of the orbits are bulged downwards. The fontanelles expand ; the sutures gape and fluctuate, the membranes beneath forming soft projecting ridges ; the eyeballs are protruded by the downward pressure of their roofs, so that during sleep a well-marked palpebral fissure is apparent. The face is small or dwarfed, and emaciated, and retains its infantile type ; this being obvious, not only from want of development, but by contrast with the expanded skull above. The scalp and subjacent bones are thin and attenuated ; the superficial veins are engorged, and readily show through a transparent skin. After a time the membranes covering the gaping sutures and spaces may become converted into bone ; or they develop adventitious platelets of bone, an attempt as it were at repair. But true ossification is rare.

As the intracranial pressure increases, the head still enlarges ; the protruding membranes become alternately tense

and relaxed during inspiration and expiration ; the integuments are tightly stretched, elevating the upper eyelid ; the eyeballs protrude ; nystagmus is frequently present ; emaciation takes place, although food is often taken greedily ; and eventually the child assumes the recumbent position, owing to difficulty in supporting the weight of its head.

In the later stages the effects of pressure are seen in convulsions, impairment of vision and hearing, partial paralyses (squints, &c.) and descending degenerative lesions of the cord, with consequent rigidities, increased reflexes, and, ultimately, atrophy of muscles. Occasionally the fluid may partially discharge itself through the nasal sutures.

The expression of face has always been stupid and heavy from the first ; and in the later stages the patient is apt to become a confirmed idiot, or to be fretful or morose in character. Notwithstanding, a hydrocephalic child under our care at the present time is fairly intelligent, and appears more cheerful and affectionate than his mates.

Prognosis.—The disease is almost invariably a fatal one. The child seldom survives to puberty, death generally occurring in two or three years at the outside. Cases, however, have been recorded which have survived to manhood.

Diagnosis.—FROM RICKETS.—Although this disease is attended by enlargement of the head, there is not that great disproportion between the sizes of the vault of the skull and the face, which is so manifest a characteristic of hydrocephalus. In rickets also, the cranial enlargements are local, and not in the form of a symmetrical, globular skull ; we should, besides, be guided by the enlarged tender epiphyses of the long bones, and by the depressed fontanelles, as compared with raised or bulging openings in hydrocephalus.

Treatment.—Usually unsatisfactory. Mercury in small long-continued doses has been recommended, but it appears to be too debilitating. Iodide of potassium has also been advocated, on the grounds of its absorbent action.¹ Bandaging the skull has been tried with indifferent success. It is liable

¹ R. Liq : Hydrarg : Perchlor : $\text{m} \times$. ; Potassii Iodidi gr. ss. ; Aquæ Camph : ad ʒj . Misce.

to cause death, by increasing the internal pressure, which we should endeavour to avoid. It is therefore, in our opinion, not justifiable. Nor have we seen any good results from blisters and other counter-irritants.

Aspiration holds out the best hopes. It should be performed with a small trocar, through the anterior fontanelle, about an inch away from the middle line. Remove only a small quantity of fluid. Avoid secondary inflammatory action by placing the patient in a cool room. A light bandage should be placed round the head, subsequent to the operation, which is not in itself dangerous, and is attended with great relief.

Improve the patient's health with cod-liver oil or vinum ferri, and attend to the digestive organs.

HEMIPLEGIA

Definition.—Paralysis of one side of the body. It is a symptom rather than a disease.

Causation.—Disease of one cerebral hemisphere. In ninety per cent. of cases the lesion is situated in the corpus striatum, or between it and the cortical centres. The most frequent cause is *hæmorrhage* ; but embolism, tumour, abscess, or other gross lesion may also give rise to hemiplegia. As regards *age* and *sex*, most cases occur in men between forty and sixty, a period when rupture of blood-vessels is most liable to happen. (See Cerebral Hæmorrhage.)

Pathology.—Usually the lesion will be found in the neighbourhood of the corpus striatum or optic thalamus ; but pressure appears to be a necessary additional factor. A clot on the cerebral surface may occur without its causing hemiplegia ; but whether the lesion be a hæmorrhage of the centrum ovale, or abscess, or an embolism, the effects which it produces are those of pressure, and therefore paralysis. So long as the cerebral fibres can carry on their functions paralysis will not result.

After a time the effects on the nerve fibres themselves are seen in degenerative changes which follow down certain well-defined anatomical tracts in the crus, pons, medulla, and cord, or which pass upwards towards the sensory cortex.

In functional hemiplegia there is no pathological lesion.

Although hæmorrhage is the most frequent pathological condition giving rise to hemiplegia, it will generally be found that there is a precedent disease of the blood-vessels, such as the changes due to senility, or the vascular lesions found in syphilis or Bright's disease ; or the results of embolism or of thrombosis.

Symptoms.—Hemiplegia is usually described as a unilateral palsy ; but this term is not absolutely correct, since ordinarily the muscles of the eyeballs, of the trunk, and the diaphragm escape. The muscles which are paralysed are those of the face, tongue, arm, and leg on the side opposite to the lesion ; those organs supplied by the third, fourth, and sixth cranial nerves being seldom affected. Loss of movement is the first symptom observed ; and although it may be accompanied by loss of sensation (hemianæsthesia), this latter sign is not necessary ; nor, if present, is it so marked as motor paralysis ; and not infrequently sensation is recovered in a short time. This may be explained on anatomical and pathological data.

The motor fibres from the cortex occupy the anterior two-thirds of the posterior division of the internal capsule, the sensory fibres being grouped in its hinder third. Since hæmorrhage from a branch of the middle cerebral artery is the most usual lesion, it follows, from the anatomical distribution of that vessel, that the motor fibres going to the tongue, lips, face, arm, and leg would be involved in that respective order ; and hence that the fibres going to the muscles of the trunk, and the fibres of sensation, escape. Further, it is possible in embolism of the middle cerebral artery, a lesion which is not so far reaching in its effects as hæmorrhage, that the nerve fibres of the tongue, lips, and face, and perhaps arm, alone may be implicated. Similarly hæmorrhage or embolism occurring in relation with the posterior cerebral artery would be indicated by the preponderance of hemianæsthesia over hemiplegia (see diagram, p. 573).

Paralysis of the face is important in diagnosis. It occurs on the same side as the affected arm and leg, but involves

only the muscles of the lower portion, those of the eyebrow and forehead escaping ; hence the patient can shut both eyes.

The tongue is also palsied on the same side as the arm and leg. This is shown in protrusion, when the tongue is pushed over by the muscles of the sound towards the paralytic side, *i.e.* away from the lesion. This action may be compared to a one-armed man pushing a wheelbarrow, which would tend to gyrate towards the armless side. The tongue when withdrawn is again pulled over to the sound side. Control over the sphincters is usually maintained. Other effects of hemiplegia are seen in vaso-motor paralysis, the temperature of the paralysed side being increased. Trophic changes, such as bedsores, œdema, and joint lesions, may also supervene.

Rigidities with increased tendon reflexes may occur, either at the onset (initial rigidity), caused by the irritation of the lesion ; or shortly afterwards, as a result of inflammation (early rigidity) ; or subsequently produced by descending degeneration involving the crossed pyramidal tract of the cord (late rigidity).

The condition of a patient suffering from hemiplegia, therefore, would be as follows. The face on the palsied side would be smooth and expressionless except in the region of the eyelids and forehead ; the eyes may be turned away from the paralysed side (conjugate deviation) ; the mouth would be drawn upwards and outwards towards the unaffected side ; the patient cannot whistle ; when asked to grin, this expression occurs on the healthy side only, and is generally exaggerated ; his sense of smell is slightly impaired from palsy of the dilator muscles of the nostrils ; the tongue is protruded to the paralytic side, the tip not corresponding with the fissure between the central incisor teeth ; and his speech (utterance) therefore is often guttural or otherwise impaired. His palsied arm lies helpless at his side, or may be suspended in front of him by the thumb being hooked inside his vest ; and his leg is also helpless, though not to such a degree as the arm, but the patient could not stand though he tried. If the injury is so serious as to be beyond recovery, and occurs in childhood, the

paralytic limbs become wasted and stunted, and the intellect shows some impairment.

Frequently, however, recovery takes place, either partial or complete. The leg shows signs of improvement about the seventh day, and before the arm. The latter limb indeed may never recover. Various theories have been advanced to account for this. Broadbent's explanation is most usually accepted. He argues that certain muscles of the body act only in unison with their corresponding opposite fellows, producing automatonism as it were (as in the eyes and legs); and that when those muscles which are set in movement by the (say) right cortex are cut off by a lesion in the internal capsule, they can still be set in motion by stimuli originating in the left cortex, and passing across by means of free commissural fibres existing between the nuclei of the two sides in the cord. For example, we have not two legs so much as one pair, and consequently one leg is no more use to us than one blade of a scissors or of a forceps; whilst we do use a left or a right arm singly, according to desire. Horsley explains this entirely on the ground of anatomical arrangement of the motor fibres in the internal capsule; and hence the lower limb fibres, being furthest from the effects of a lenticulo-striate hæmorrhage, are affected latest, and are also the first to recover their function.

But even this appears quite consistent with Broadbent's view, and the axiom might read thus: 'Those fibres controlling automatic muscular actions are furthest removed from the site of most probable injury; and there is free intercommunication between the nuclei for these actions.'

The muscles therefore which escape in whole or part are those of the eyeball (third, fourth, and sixth nerves); those concerned in mastication (fifth); the muscles of phonation (vagus), and the trunk and legs.

There are, in addition, certain special symptoms which are peculiar to lesions in certain localities. Thus, if the leg, or face, or tongue, or any part which is usually involved in hemiplegia be unaffected, we may suppose that the lesion is a small one, and situated between the cortex and the congru-

gated fibres of corpus striatum and involving only certain tracts ; in other words, the nearer the lesion is to the cortex, the more likely is the lesion to be partial. But even cases of monoplegia due to lesion of the internal capsule have been recorded, although they are very rare.

If the lesion extends from the left corpus striatum to the third left frontal convolution and island of Reil, it is attended by right hemiplegia and aphasia, independent of affection of the tongue muscles.

Lesion of the crus cerebri causes paralysis of the third nerve of the same side, and of the opposite side of the body (cross paralysis).

Lesion of the pons causes complete paralysis of the fifth, sixth, and seventh nerves of the same side, together with paralysis of motion and sensation of the opposite side. It is also attended by more or less fever. (See p. 579.)

Lesions of the medulla may cause paralysis of both sides of the body, the motor tracts here being approximated. Such lesions are usually fatal, from involvement of the nuclei of the vagus and other vital nerves.

Hemiplegia attended by coma is generally due to hæmorrhage.

Sudden hemiplegia without coma is generally caused by a patch rapidly softening or by an embolism.

Gradual hemiplegia is frequently due to the slow growth of a cerebral tumour.

Complete hemiplegia with impaired intelligence is generally secondary to combined hæmorrhage and softening.

In hysterical hemiplegia the loss of sensation is in excess of loss of motor power ; there is no motor paralysis of the face and tongue ; both hemiplegia and hemianæsthesia are transient ; and loss of power is usually more pronounced in the leg than in the arm.

Treatment.—Rest, both bodily and mental, is the essential point. We should also endeavour to lessen arterial tension, by venæsection if the patient is of full plethoric habit, or by bromides, or by purgatives. Iodides are required in cases of syphilitic history, and should be given in a prolonged course.

Subsequent stimulation of the paralysed muscles may be necessary, either by the help of electricity or by strychnia ; but it must not be attempted till all rigidity and signs of central irritation have disappeared.

SCLEROSIS

Definition.—Literally a ‘hardening.’ The term has of late years been entirely restricted to chronic inflammation of the brain, spinal cord, or nerves. The subject is considered in this section for convenience only.

Causation.—*Age.*—It occurs at all periods, but especially in middle and adult life. *Sex.*—Women are most frequently affected in early adult life, and men in late adult life. *Heredity* has no direct influence ; yet parents with a neuropathic tendency often beget children who eventually suffer from sclerosis or other neurosis. *Cold and Injury.*—Frequent exposures to cold are stronger predisposing factors than a sudden severe exposure : the latter is more likely to produce acute myelitis. Similarly an injury which at first appeared trivial may produce slow nutritive changes which lead to sclerosis. *Diseases.*—It may be secondary to chronic alcoholism, the inflammation beginning at the surface and extending inwards. It may be a manifestation of gout, either acquired or hereditary. Syphilis is undoubtedly a potent factor, but its influence is probably exaggerated. If combined with alcoholism, the two act as strong predisposing causes. Finally, it may be consequent on chronic inflammation of the meninges, by which a constricting pressure is exerted on the nervous matter beneath.

Pathology.—On section of a sclerotic area the white substance is grey in tint, the grey matter being darker still. In the early stages the affected part is swollen, often raised above the surface of the brain or cord, and adherent to the membranes. In the later stages the patch is denser and shrunken, and it is easily stained by logwood, carmine, and other reagents. Sclerotic changes, further, tend to involve definite tracts in the brain and cord, and to be limited thereto ; and to this pathological feature we are indebted for much knowledge as to the histology and physiology of the nervous system.

Microscopically sclerosis presents certain constant changes, viz. a great increase in the connective-tissue elements, and a destruction of nerve tubules and cells. The connective tissue is rich in inflammatory cells in the early stages, whilst in advanced states fibrillæ are more pronounced. Nevertheless, the interstitial connective tissue is not entirely fibrillar; in certain parts it becomes amorphous and structureless. The nerve tubules either waste early, probably as a result of atrophic influences, or later as a sequel of surrounding pressure; hence there is seen a trabecular network of connective tissue occasionally enclosing empty spaces once occupied by nerve tubules; or the tubules are merely represented by an axis cylinder only; or they are moniliform and otherwise distorted by unequal pressure.

The blood-vessels are also thickened, their calibres diminished, and they are embedded in an increase of interstitial tissue, the result of chronic inflammatory changes extending from the capillaries themselves.

In the grey matter the interstitial tissue is also exaggerated. The multipolar ganglion cells become at first enlarged; they then shrink, lose their processes, and become granular, pigmented, and finally disappear.

In a given patch of sclerosis all the above changes may be seen at the same time. Three zones are recognised; in the innermost the changes are most advanced; in the outermost the changes are in an early stage only; the middle one being also intermediate in the extent of its histological alteration. Bearing this in mind, we expect to find in the outer circle that the tubules are little altered, except that they are pushed wide apart by the advent of the new connective tissue growth; in the intermediary part the tubules are compressed together by the shrinking of this connective tissue; they are also irregular in longitudinal outline, being constricted here and bulged out there, according to the irregular squeezings of the inflammatory tissue; whilst in the central area this inflammatory tissue is predominant, destroying the cells and tubules, so that it is practically the only tissue seen, with

the exception, perhaps, of an axis cylinder or a nerve-cell here and there to denote their former existence.

A sclerotic area, wherever situated, is better distinguished from the surrounding healthy tissue by the naked eye than by the microscope.

GLOSSO-LABIO-LARYNGEAL PALSY (BULBAR PARALYSIS)

Definition.—A progressive form of paralysis, due to lesion in the medulla oblongata, and characterised by paralysis of the muscles of the tongue, lips, and larynx or pharynx.

Causation.—The *primary* form of the disease has been attributed in many cases to syphilis or to rheumatism. In others it has supervened on severe mental shock and anxiety. *Sex.*—Women suffer more frequently than men, but authorities differ on this point. *Age.*—It is a disease of adult life.

Secondary.—Sclerotic change may invade the medulla by extensions from other parts. It frequently appears as the last stage of disseminated sclerosis, or of lateral sclerosis. Again, it may complicate, and actually be the cause of death in, locomotor ataxia.

Pathology.—The sclerotic changes are situate in the medulla, involving the motor nuclei of the hypoglossal, glosso-pharyngeal, spinal accessory, and facial nerves principally. The nuclei of these nerves, especially of the twelfth and seventh, are close together, probably on account of the concerted action of the muscles which they supply. The motor cells are shrunken, atrophied, and pigmented, their processes being lost or rendered tortuous. The nuclei of the vagi are also occasionally involved, though not to such an extent as the other medullary nerves. A similar remark applies to the motor roots of the upper cervical nerves. The degeneration is not limited to the nuclei, but also extends down the nerves originating therefrom.

Symptoms.—Seeing that the lesion is one of the medulla, involving the motor nuclei of the fifth, seventh, ninth, tenth, eleventh, twelfth, and possibly one or two of the higher cervical nerves, the principal symptoms are paralysis of muscles supplied by these nerves. The paralyses are thus somewhat

wide and complex in their distribution, but they are symmetrical. The tongue is usually first affected: the patient cannot raise it to the roof of his mouth. He has difficulty in speaking, especially in articulating such words as begin with labial and dental consonants, such as 'this,' 'to-morrow,' and all others which require 'the use of the lips and of the tip of the tongue,' the latter phrase being in itself a good test one. As the disease advances, the tongue is protruded and retracted with difficulty, and it eventually lies motionless in the floor of the mouth, pressed and indented by its own weight against the teeth.

Deglutition is performed with difficulty; saliva accumulates and dribbles from a mouth half opened, with pendulous lips, giving an expression of imbecility, which is characteristic. Or, in consequence of paralysis of the palate muscles, food returns by the nares; or passes into the larynx; or accumulates in the outer buccal cavity. Subsequently the consistency of the saliva is thickened, either by changes in the secreting apparatus itself, or owing to dryness of the mucous membrane produced by the patient's open mouth. The secretion thus hangs in sticky threads from the roof of the mouth, or from the corners of the lips. The voice itself is altered. The patient speaks in grunts, or with a leathery intonation as in myxœdema; this being brought about not only by the affection of the muscles of the tongue, lips and palate, but also of those of phonation (laryngeal). The buccinators are rarely involved; but grinding movements (pterygoids) of the jaws are performed with difficulty. Sooner or later, symptoms of affection of the vagi nuclei are apparent. The patient has dyspnœa; there is bronchial catarrh with accumulation of bronchial mucus from paresis of the bronchial muscles; the circulation becomes enfeebled; and the heart's action, frequently accelerated in the early stage, gradually gets slower and intermittent. Attacks of syncope and dyspnœa are frequent; and they may be aggravated by food which, having passed the fauces, becomes arrested in the lower pharynx and œsophagus. During the whole course of the illness there is no loss of sensation; no loss of control over the sphincters; the mind keeps

clear ; and there is no fever unless from pneumonia or other complications.

The disease is a progressive one. The patient becomes enfeebled from insufficient nourishment ; the pulse gets weaker ; he becomes depressed in spirits, and dies from asphyxia, or syncope, or from the result of food which having passed into the air-passages sets up a fatal form of broncho-pneumonia.

Diagnosis.—FROM GENERAL PARALYSIS OF THE INSANE.—This disease is distinguished by its mental condition (exaltation or depression) ; its tremors affecting the tongue, facial muscles and lips, and the irregularity of pupils with a comparative loss of response to the action of light.

Prognosis.—Always a grave disease. Death usually occurs within a twelvemonth, whether the disease be due to a primary lesion or secondary to progressive muscular atrophy. Occasionally, when caused by medullary hæmorrhage or embolism, the course of the disease is much more rapid and acute.

Treatment.—All we can do is to try to relieve dangerous and serious symptoms. Food must be given somehow, either by stomach pump or by enemata. We have no experience of any relief from galvanism, which has been recommended.

DISSEMINATED SCLEROSIS (INSULAR OR MULTIPLE SCLEROSIS)

Definition.—A chronic degenerative disease, due to patches of sclerosis scattered throughout the brain and central nervous system, and characterised by tremors of muscles during their action, nystagmus, a ‘scanning’ speech, and eventually by the onset of paralysis.

Causation.—*Age.*—Most frequent in youth and early adult life ; rare after thirty. *Sex.*—Commoner in women. *Heredity.*—Often a strongly-marked feature, independent of any neuropathic tendency, such as epilepsy and the like.

It is also believed to be induced by the influence of repeated cold and exposure.

Pathology.—The sclerotic patches may occur on the surface of the brain, especially its base, in the pons, medulla, or spinal cord. They are sharply defined, somewhat dense, and

adherent to the overlapping membranes. They vary in size from large shot to an acorn, and appear to invade the white matter to a larger extent than the grey. The cranial and spinal nerves may even be the subjects of the indurated change.

Microscopically a patch would present an increase of the neuroglia, in the form of a delicate fibrous tissue compressing and distorting the nerve tubules, or even entirely destroying them. In the more advanced parts the nerve tubules may be entirely replaced by the new tissue formation; or, at least, only an axis cylinder here and there denotes the previous histological character of the part. The capillary walls are also thickened, and surrounded by proliferated inflammatory nuclei.

Symptoms.—It is obvious that the symptoms are very various and complex, depending as they do on the site and extent of the sclerotic patches. The following, however, are usually present, viz. : rhythmical tremors of muscles, occurring only during exertion and ceasing during rest ; paresis of muscles, usually beginning in one leg ; a scanning, syllabic utterance ; nystagmus ; gyratory vertigo ; and mental impairment.

The tremors occur only when muscles are brought into action. The patient thus drinks with difficulty ; the cup in its course to the mouth, although it eventually arrives there, is somewhat violently shaken, and its contents often spilt (see fig. 29) ; when asked to sit on the side of his bed and take his feet from the ground, his trunk oscillates and his arms are stretched out to act as a balancing pole. Similar tremors affect the head and lower limbs. But they are not necessarily universal ; they may be limited to one leg, or arm, or to the trunk muscles. Occasionally they do not supervene until the disease is tolerably advanced. Muscular paresis is also present. It usually commences in one leg and then extends to the opposite one. Subsequently the arms become affected ; and eventually there is complete paralysis in both upper and lower limbs. Notwithstanding this, the muscles usually retain their bulk, tonicity, and muscular sense. The patient is not ataxic ; and he retains control over bladder and rectum.

The speech is characteristic ; the lips are tremulous. He

is slow and deliberate in his utterances. Each word appears to be broken up into its component syllables, and he appears to mentally place a comma or pause between words. Nystagmus is a variety of rhythmical tremor. It is easily induced by the eye being fixed on some object (say the observer's pen) which is then moved laterally or vertically. This symptom is absent during sleep. There is often, in addition, a form of amaurosis due to atrophy of the optic disc.

Vertigo is common. It is generally gyratory in character, being also referred to objects which are being looked at, and is apparently aggravated by the movements of the eyeballs. Mental impairment comes on sooner or later. The patient becomes forgetful ; he is emotional, or is unnaturally cheerful and silly ; or he is suspicious, and possibly imbecile or maniacal.

Eventually attacks of rigidity occur in the lower extremities. The limbs are from time to time rigidly set in the position of marked extension, the attacks lasting for some hours or days, and separated by periods of comparatively normal condition ('spinal epilepsy'). Subsequently, however, the rigidities become permanent, and a similar condition is found in the arms. The rigidities are occasionally accompanied by violent tremors of the muscles, easily induced by cold, tickling, or other form of stimulus. The deep reflexes are increased.

The last stage is marked by apoplectiform attacks, accompanied by sudden rise of temperature, by diminishing mental powers, by loss of control over the sphincters, with chronic cystitis and bulbar complications.

It must be remembered, however, that it is not every case of disseminated sclerosis which presents the above chain of symptoms. Much will depend on the extent and locality of the cerebral and of the spinal lesions. So far as the cord is concerned, many cases bear a resemblance to locomotor ataxy, through involvement of the posterior root zones ; others have symptoms pointing to degeneration of the lateral tracts ; whilst, again, others suggest the invasion of the anterior horns of grey matter. The presence of scanning speech, muscular tremors and nystagmus, however, will point out the true nature of the disease.

Prognosis.—Always a fatal disease. The gravity of the attack depends on the extent of the sclerotic affection. Life may be prolonged for ten or twelve years ; but a speedier termination of the disease may be expected if the brain and spinal cord are both involved.

Diagnosis.—The disease may be confounded with Paralysis Agitans, Chorea, Chronic Alcoholism, and Cerebellar Tumour. The distinctive features appear in the following table :

—	Paralysis Agitans	Chorea	Chronic Alcoholism	Cerebellar Tumour
Tremors . .	Not so coarse, and occur even during muscular rest	Movements spasmodic, violent, purposeless, and not rhythmic	Tremors are accompanied by muscular twitchings. They cease during abstinence from alcohol	Rare, except in late stages during a sustained effort
Age . . .	Advanced life .	Youth . . .	Middle age .	Youth
Nystagmus .	Not present .	Not present .	Not present .	Often present ?
Convulsions .	No true convulsions	Spasmodic .	In last stage .	Present from onset
Vertigo . .	None . . .	None . . .	None . . .	Often present
Vomiting . .	Not present .	Not present .	Occasionally present	A marked symptom
Mental symptoms	None, till late stage	Only mental dulness	Busy delirium	Occasionally
Speech . . .	Not affected .	Not affected .	Incoherent .	Not usually affected
Optic neuritis .	None . . .	None . . .	Occasionally .	Always

Treatment.—Nothing avails medicinally, so far as we know. Strychnia is the only drug which has afforded even temporary relief.

GENERAL PARALYSIS OF THE INSANE

Definition.—A form of madness, characterised by delusions, and attended by tremors of muscles especially of the face and lips.

Causation.—*Age.*—The disease occurs almost entirely during the prime of life. It is seldom seen before forty or after sixty years of age. *Sex.*—Males predominate in the proportion of quite four to one. *Nervous Influences.*—In nearly all instances there is a history of either business worry, alcoholism, or of sexual excesses. Even success in business or occupation will, in some instances, predispose to the disease.

Pathology.—The morbid changes are practically such as would appear in chronic inflammation of the cortex of the brain and of its membranes. The cranial bones are even thickened ; there is a more or less diffuse pachymeningitis, with adhesion of the membranes especially to the frontal and upper surfaces ; the arachnoid fluid is thick and turbid, or occasionally purulent ; the intraventricular fluid has the same characters ; the cortex is deeply engorged with blood ; perivascular extravasations are found ; the nerve fibres show degenerative changes ; and the cortical nerve cells, especially the branching pyramidal set, become attenuated and pigmented.

Similar changes may be observed in the spinal cord and its envelopes. The bulk of the cord itself is shrunken and wasted ; and sclerotic changes are found in the posterior external or in the lateral columns.

Symptoms may be divided, as the name of the disease implies, into two chief groups, namely (*a*) mental and (*b*) paralytic. There is, however, no order of onset of the symptoms. In one case paralytic affections may precede the mental failure ; in another a sudden outbreak of madness is the first recognised symptom.

The history of a case is often somewhat as follows. A young robust man, of previous good health, suddenly shows mental failure. He loses his memory, or neglects his business, or launches out recklessly into expenditure beyond his income. Then when medical advice is sought, it is found that he has also difficulty in articulation, that he is perhaps ataxic in his gait, that the pupils are unequal, that he is emotional, that he has exaltations, and that his usual shrewdness and good judgment have left him. A more detailed description, however, is necessary.

(*a*) *Mental Symptoms.*—The patient has characteristic exaltations. This condition certainly may be, in some instances, preceded by severe mental depression. He imagines that he has committed some terrible sin or crime, or that he has lost every penny of his savings. According to some authors this depression stage constitutes a distinct variety, since it may last throughout the course of the disease. In most cases,

however, it would appear that it is a state only prior to that of exaltation, during which he imagines that he is indispensable to the State, or that he is an emperor, or that he himself, or his sons and daughters are the handsomest of their sexes. He does not appear to know the true value of words. He imagines that he is a giant in intellect or in strength ; or he airily writes a cheque for a million sterling. He purchases luxuries wholesale, and yet is niggardly in buying necessities. At the same time he is restless in manner, and apparently is never tired ; yet he cannot carry on a train of thought. His moral character also becomes perverted ; he is emotional or passionate ; he may be indecent in speech or gesture, or he may steal, or even commit homicide or other serious crime.

(b) *Paralytic Symptoms*.—The characteristic symptom under this heading is tremor, of the lips, tongue, and face during movement. His speech therefore is blurred, dreamy, or hesitating. He has great difficulty with such words as contain many ‘labials.’ Good test-words to bring out this symptom are ‘biblical commentary’ and ‘royal artillery’ (Savage). Facile movements of the fingers, such as are required in writing or piano-playing, are lost. Occasionally this failure would appear to exert some influence on the handwriting, which previously illegible, on account of carelessness and rapidity of movement, now becomes laboured and legible.

The expression of the face is fixed, staring, and gloomy.

The muscular sense is impaired, so that differences of weight between two objects are not recognised. The gait is often distinctly ataxic ; and there is atrophy of the optic disc. The knee-jerks may be increased, or entirely lost. The temperature is usually raised to between 100° and 102° ; and, according to Savage, the skin is often abnormally greasy. As the disease progresses, the patient is liable to epileptiform or to apoplectiform seizures. Or, perhaps previously quiet and harmless in his exaltations, he suddenly becomes maniacal, when he may injure himself or his dearest friend, or may set fire to his house, or perpetrate some equally terrible deed.

In the last stage of the disease the delusions go ; indeed,

his mental power disappears altogether. He knows nothing that goes on around him ; he passes his evacuations in bed, and is apparently only kept alive by the medullary centres remaining intact. Finally these fail, and the patient sinks exhausted after some convulsions, or he dies from asphyxia caused either by choking, or from interference with the functions of the vagi nerves. Often cystitis or bedsores complicate the last stage of the disease.

Varieties.—Certain varieties of general paralysis have been described according as the different symptoms predominate. Authors thus speak of the expansive (described above), the melancholic, and the maniacal varieties. Again, a group of cases is characterised by tabetic symptoms, with marked facial pallor ; in another group with flushed countenance, the knee-jerks may be increased, with symptoms resembling lateral sclerosis. Yet another group is characterised by symptoms which are indistinguishable, except for the mental failure, from disseminated sclerosis.

Treatment.—No medicinal treatment appears to be of the least avail. It is recommended that the patient be removed from all anxiety and worry. This often entails a burden on the patient's means which can ill be borne, and probably life is very little prolonged by it. It is, however, absolutely necessary that the patient should be under proper care and supervision, as he is liable at any moment to sudden maniacal outbursts.

LESIONS OF CRANIAL NERVES

The FIRST, or OLFACTORY NERVES, leave the olfactory bulb and pass to the upper part of the nasal fossa, through the foramina in the cribriform plate of the ethmoid. The filaments consist of non-medullated tubules. Fibres have been traced backwards to the uncinate gyrus of the temporal lobe ; some others pass by the anterior commissure to reach the cortical centre of the opposite side.

This nerve may be injured at the bulb, or it may be implicated at its distribution.

Loss of smell (*anosmia*) may occur from lesions of the

centre, injury to the bulb, or from affections of the mucous membrane of the nose, and also from paralysis of the muscles of the nostrils. The sense of taste is frequently lost or impaired at the same time. Various subjective olfactory sensations occur in hysteria and in insanity. An epileptic aura may take the form of some pungent or unpleasent odour. *Hyperosmia*, or increased sensitiveness to odours, may also occur in hysterical women and in hypochondriasis. Tests which are used should consist of some of the aromatic oils. But musk is perhaps the most delicate test. Pungent vapours must be avoided.

Second, or Optic Nerve

The centre for vision is in the occipital lobe, especially in the cuneate lobule. From this region the fibres, which are called the optic radiation, pass forward to the posterior part of the internal capsule, where they meet with fibres coming from the opposite side of the brain through the corpora quadrigemina, the corpora geniculata, and the optic thalamus. The fibres thence proceed as a large bundle, the optic tract, which embraces, or winds round from without inwards, the crus cerebri and is also attached to it ; then, meeting its fellow from the opposite side, at the base of the brain they unite to form the optic chiasma or commissure. From this commissure the optic nerves diverge, and, running forward, each enters its orbit through its optic foramen, carrying with it a sheath of dura mater ; finally the nerve pierces the sclerotic to the inner side of the middle line of the eyeball, and is distributed to the retina.

At the chiasma only certain fibres from the optic tract decussate with their fellows, viz. those which supply the inner halves of the two retinæ ; whilst those fibres which supply the outer half of a retina are contained in the outer layer of the optic tract of the same side. In other words, the right tract supplies the right side of both eyes, and the left tract the left side of both eyes (see fig. 30, p. 652).

A third set of commissural fibres runs from tract to tract at the back of the chiasma, but these are not concerned in vision.

The OPTIC NERVE may be the subject of Inflammation (optic neuritis) and Atrophy.

The **causes** of optic neuritis may be divided into local and constitutional.

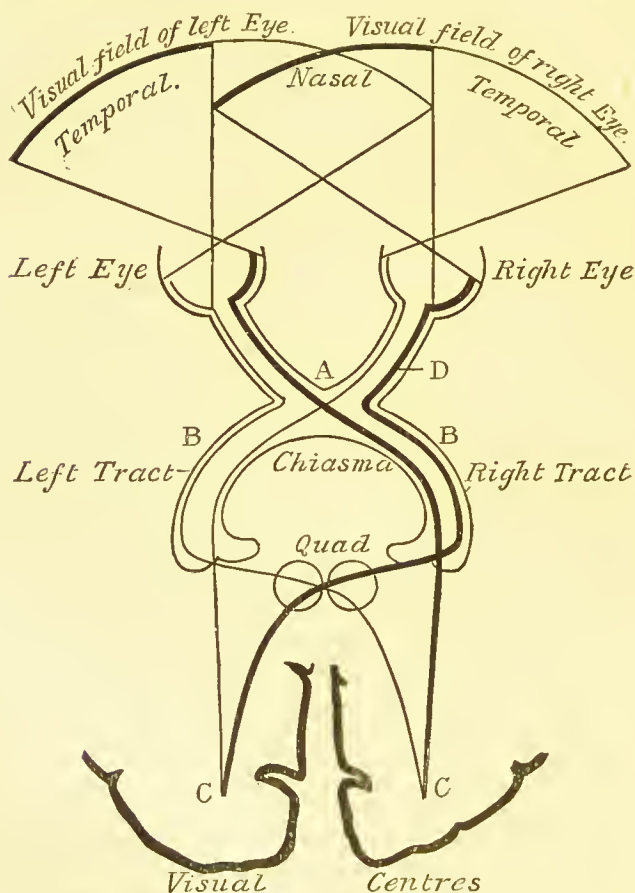


FIG. 30.—DIAGRAM OF OPTIC AND VISUAL TRACTS, MODIFIED FROM CHARCOT AND FROM STARR

The *local* causes, which are generally coarse lesions, may originate in the orbit or within the eranium. They consist, for the most part, of tumours, such as tuberele, sarcoma, aneurysm ; or inflammatory thickenings of the meninges ; or

hydrocephalus. They cause neuritis principally by their pressure effects ; and obviously, therefore, the neuritis may be unilateral or bilateral. In meningitis and other cerebral lesions the neuritis is always bilateral ; in orbital disease, especially when the lesion is close to the sclerotic, the affection is usually limited to the eye affected.

The principal *constitutional* causes are, Bright's disease, leucocythæmia, lead poisoning, chlorosis, syphilis, and influenza.

On examination with the ophthalmoscope the disc is seen to be red and congested, the edges blurred, and the veins tortuous ; the arteries are not much altered. As the inflammation advances, the disc becomes swollen, large, prominent, and striated. Subsequently it presents a mottled or patchy appearance, from effusion of lymph or blood. These conditions may abate and leave no change in the optic nerve ; but in severe cases the acute inflammatory processes only subside very slowly, and leave a condition known as Optic Atrophy, in which the disc is shrunken and opaque, with sharply cut or irregular edges and small vessels. The retina is often also involved at the same time (neuro-retinitis).

Optic atrophy, although usually a condition secondary to neuritis, may be a primary affection, as in locomotor ataxia.

Symptoms of optic neuritis are, chiefly, contracted field of vision, and dimness of sight. The disease, however, may exist in a mild, or even marked degree, with no impairment of sight.

In optic atrophy the symptoms usually observed are dimness, reduced, or altered field of vision, and altered perception of colour.

In both conditions the physical signs as revealed by the ophthalmoscope, outweigh the symptoms.

The OPTIC CHIASMA, TRACT, and VISUAL CENTRE may be implicated by similar lesions as those which affect the nerve in front of them. They produce special symptoms according to their extent and situation. A lesion of the optic chiasma produces symptoms which vary in intensity according to the extent of the injury. Thus :

(1) A superficial injury at A (fig. 30) produces blindness of the temporal portions of each field, since only the inner

decussating or nasal portion of the retinal fibres would be involved (temporal hemianopia).

(2) A more extensive lesion may implicate the entire fibres of one tract, as well as the decussating fibres, and so produce total blindness in one eye, and temporal hemianopia in the other.

(3) A gross lesion may involve chiasma and both tracts, producing total blindness.

(4) A lesion affecting the outer fibres of one tract (B) produces loss of vision in the nasal half of the field, by paralyzing the temporal side of the retina only. If a similar lesion exist in both tracts, both eyes would, of course, be equally involved (nasal hemianopia); but this latter condition is rare.

(5) A lesion involving the entire fibres of one tract, say the right, produces loss of visual function in the temporal half of the right eye and in the nasal half of the left; so that the patient cannot see objects on his left side (homonymous hemianopia). He therefore turns his head to the blind left side, so as to receive direct central rays on the normal half of each retina.

(6) A destructive lesion at one cortical eye centre (c) produces total blindness of the opposite eye; a similar result on the same side ensues from destruction of one optic nerve (d).

Many, or indeed all, of the above symptoms may be functional only, as in hysteria and megrim.

The Third, or Motor Oculi Nerve,

arises from grey matter in the floor of the aqueduct of Sylvius, beneath the nates. From this point the fibres pass through the red nucleus of the crus, and appear at the surface as a rounded nerve at the inner border of the crus. It pierces the dura mater between the anterior and posterior clinoid processes, and entering the outer wall of the cavernous sinus, it passes to the orbit through the sphenoidal fissure to supply all the muscles of the eyeball; except the external rectus and the superior oblique. This nerve has not yet been traced to the motor cortex.

Causation.—As in the other forms of cranial nerve para-

lysis, it may be caused by rheumatic or syphilitic thickenings of the nerve or of the membranes which it pierces. Occasionally it is found as a sequel of diphtheria. It may also be directly caused by any form of tumour (new growth, gumma, hydatid cyst, hæmorrhage) situated near and involving the crus cerebri, thus causing crossed paralysis; or the disease may be situated in the aqueduct of Sylvius, or at the nucleus of nerve, when a general ophthalmoplegia occurs, owing to the centres for the other eye muscles being involved; finally, a lesion may occur in the sphenoidal fissure, or in the orbit. Should paralysis of the third nerve occur, with hemiplegia of the same side, it cannot be caused by a single lesion. Lastly, the paralysis may be partial, one branch or division only being affected, when the lesion is probably situated in the orbit.

Symptoms will vary according as the whole nerve is paralysed or a portion only. In complete paralysis there is drooping of the upper eyelid, the eye is turned downwards and outwards by the superior oblique (fourth) and the external rectus (sixth). The pupil is fixed and dilated, or remains in a position midway between contraction and dilatation owing to the elasticity of the iris.

When one rectus alone is involved (the inferior oblique is very rarely affected by itself) there is strabismus (the eye being rotated towards the opposing healthy muscle) and diplopia, the distance between the false and the true images increasing as the object is moved in the direction of the action of the palsied muscle.

But it is rare for single muscles to be affected, or, at least, completely paralysed; and hence, as the movements of the eye are very complex, every one being brought about by more than one muscle, it is difficult to apportion the exact amount of paralysis to each muscle.

Abduction by the external rectus alone (sixth nerve) is probably the only exception to this rule.

For more exact details the reader should consult Ross, Gowers, and other authors.

Treatment depends on the cause. In rheumatic or

gouty subjects prescribe alkalies or salicylates, combined with iodide of potassium. If there is a syphilitic history our chief hope is in the iodides of potassium or of sodium for some continued time. Apply eserin to the eye ($\frac{1}{2}$ gr. to 3j.) when the sphincter fibres of the pupils are paralysed.

In the paralysis due to diphtheria, iron and strychnia are indicated.

Galvanism (voltaic current) may be tried to the affected muscles.

If the condition be due to intraorbital tumour, the question of surgical operation will have to be considered.

The Fourth Nerve

rises from the valve of Vieussens and decussates therein with its fellow. Its fibres have been traced through the grey matter lining the aqueduct of Sylvius to a nucleus beneath the testes, but as yet its connection with the motor convolutions has not been defined. The nerve pierces the dura mater behind and external to the posterior clinoid process, and passing along the outer wall of the cavernous sinus, enters the orbit by the sphenoidal fissure, to be distributed to the upper surface of the superior oblique.

This nerve may be implicated at its nucleus in the upper part of the fourth ventricle by tumour, hæmorrhage, and degenerative changes, in common with the nuclei of the third and sixth nerves.

The **symptoms** consist of defective downward and inward movements of the eye, accompanied by double vision when the patient looks down.

The Fifth Nerve

is composed of a large sensory portion, on which is the Gasserian ganglion, and a smaller or motor portion.

The deep origin of the nerve is from the grey matter in the floor of the aqueduct of Sylvius, external to the nucleus of the third nerve. There are two sensory nuclei: an upper one, extending from almost the anterior end of the aqueduct to the middle part of the bulb, and a lower one from immediately below the termination of the first down to the

upper part of the cord. The motor nucleus is internal to these, at about the same level where the upper and the lower sensory nuclei meet.

Its motor cortical centre is in the lower third of the ascending frontal convolution. The sensory part of the nerve issuing from the Gasserian ganglion divides into three portions : the ophthalmic, which enters the orbit through the sphenoidal fissure ; the maxillary, which passes out through the foramen rotundum ; and the mandibular, which emerges through the foramen ovale. With this third portion the motor root joins immediately after their exit through the foramen ovale.

Causation.—Injury to, or pressure upon the nerve at its nucleus or in some part of its course. Pressure is the usual exciting cause, and it may be exerted by many forms of new growth, by syphilitic gumma, by abscess, by hæmorrhage, or by hydatid cyst. The lesion may occur at the nucleus of the nerve in the upper extremity of the medulla ; at the pons Varolii ; at the point where it runs beneath or perforates the dura mater ; or in any of the three great divisions after the Gasserian ganglion.

Symptoms will vary according as the injury is situated before the Gasserian ganglion, and involving both motor and sensory roots, or after the ganglion, and affecting one or other of the three primary divisions.

If the *first* division alone be involved, the symptoms are mainly loss of sensation to the upper eyelid and conjunctiva (lachrymal), to the forehead (frontal), to the side and tip of the nose, and to the mucous membrane of the lower part of septum nasi (nasal). The cornea becomes opaque and sloughs, the whole eye becoming ultimately disorganised if the Gasserian ganglion is implicated.

When the *second* division alone is affected there is a similar anæsthesia in the lower lid and conjunctiva (palpebral), in the teeth of the upper jaw (anterior and posterior dental), and in the upper lip (labial), the ala of the nose (nasal), and in the soft palate.

In affection of the *third* division there would be paralysis of the muscles of mastication. The chewing movements of

crushing and grinding would be weakened, and to some extent there would be difficulty in opening the jaws (external pterygoid) ; but this latter movement could be carried on to a great extent by the muscles attached to the hyoid bone and the lower maxilla. In addition, protrusion being performed by the healthy muscles, the jaw would be rotated towards the paralysed side, whilst retraction would similarly be somewhat rotatory in character towards the healthy side.

Food would collect, without knowledge, in the cheek, by reason of sensation being lost to the buccinator. There would be unilateral anaesthesia and dryness of the tongue and gums. A similar affection would involve the skin of the face from behind the distribution of the superior maxillary nerve terminations, as far back as the parotid gland and the external auditory meatus (auriculo-temporal).

If the nucleus, or the superficial roots be injured, all the above symptoms would be combined, although it is conceivable that multiple lesions might pick out various branches of distribution in all three primary subdivisions. The disease is generally unilateral.

Treatment.—Entirely dependent on the cause. Iodide of potassium and mercury are indicated when there is a syphilitic history. We may advise operation if the lesion be within the reach of surgical means and the case be otherwise suitable. In rheumatic or gouty patients the best hope lies in treating the disease of which the paralysis may be a part. Galvanism should be applied to the affected muscles.

The Sixth Nerve

pierces the dura mater above the fusion of the occipital and sphenoid bones, then enters the floor of the sphenoidal sinus, and appears in the orbit through the sphenoidal fissure, to supply the external rectus.

The nucleus is almost in the centre of the floor of the medulla, quite close to the middle line. A bundle of fibres runs from this nucleus, and decussates below the corpora quadrigemina with a similar set from the opposite side, and afterwards joining the third nerve is distributed to the internal

rectus. Hence the eyes can be directed either to the right by the influence of one centre, or to the left by the opposite centre.

Paralysis of this nerve, usually caused by basic meningitis, or by gumma, is characterised by internal squint, and by diplopia on looking towards the affected side.

According to Beever, affection of the nerve at its nucleus also involves those fibres which pass to that part of the nucleus of the opposite third nerve which supplies the internal rectus, so that the other eye cannot be turned inwards, and consequently both eyes are conjugately deviated to the opposite side, away from the lesion.

The Seventh, or Facial Nerve,

enters the internal auditory foramen with the auditory nerve, then passes by itself through the aqueduct of Fallopius, and emerges at the stylo-mastoid foramen. The nucleus is in the floor of the fourth ventricle, about the same latitude as the sixth nerve, and the same longitude as the fifth, but deepest of all.

FACIAL PARALYSIS (BELL'S PALSY)

Causation.—Lesion to the nerve may occur—(1) within the cranium, either at its cortical centre (lower end of ascending frontal and ascending parietal convolutions); or between the cortex and the nerve's nucleus in the medulla; or at its nucleus; or at its superficial origin at the outer side of the olivary body; or between its superficial origin and the internal auditory meatus. (2) Within the aquæductus Fallopii. (3) After its emergence from the stylo-mastoid foramen.

Within the cranium it may be caused by hæmorrhage, new growth, syphilitic gumma, tubercle, or by meningitis.

Within the bony canal it is usually due to neuritis or effusion caused by syphilis, rheumatism, caries of bone, or fracture.

Externally to the skull it may result from cold, blows or injury (as in forceps delivery), and parotid tumours. Frequently there is no discoverable cause.

Symptoms.—There is most palpable difference between the

two sides of the face : the affected side is smooth, motionless, and expressionless, whilst the other side is unaltered ; or the furrows and lines may even be exaggerated. Thus the angle of the mouth on the palsied side is depressed, the half of the lower lip drooping ; the patient cannot whistle, or at least the attempt is only at one side ; when asked to grin and show his teeth, he exposes those on the sound side only ; food collects between the buccinator and the teeth. He cannot wink ; the expression of the eye is fixed and staring, as though he wore an artificial glass-eye ; tears suffuse the conjunctivæ and overflow the lids ; conjunctivitis may occur owing to exposure, and to incomplete sweeping of the front of the eye by the lids. The conjunctival reflex is absent. Half the forehead is smooth, and entirely without the expressions of surprise (*occipito-frontalis*), frown, or suffering (*corrugator supercilii*). The nostril is less widened, and does not dilate on sniffing or deep inspiration, thus impairing the sense of smell.

The arch of the palate may be lower on the affected side (*levator palati*), and the uvula may be drawn to the opposite side (*azygos uvulæ*). The mouth may be dry, and the tongue tasteless on the side involved (*chorda tympani*).

The patient may also complain of 'drummings' and other noises in the ear (*paralysis of laxator tympani*) ; or he may be completely deaf if the auditory nerve be also involved.

The palsied muscles at first show an increased irritability to the faradic current, but soon lose it entirely, whilst retaining their galvanic irritability.

Therefore all the external branches of the seventh nerve are implicated, and the paralysis is 'complete.' But there is no anæsthesia. Eventually there may ensue various secondary distortions from contraction of separate muscles.

Diagnosis.—In HEMIPLEGIA, examination would reveal loss of power extending completely to one side, upper and lower extremities being both affected. In addition there would be paralysis of the tongue, and of the muscles of mastication on the same side ; whilst the facial paralysis, though evident, is not so complete as in Bell's palsy, and does not extend to the muscles of the eyelid and forehead, which are

supplied by the temporo-facial division of the seventh nerve. This is probably owing to associated expression-movements of the two sides.

SITUATION OF LESION.—If on the side affected there is also internal squint, the lesion is probably in the fourth ventricle, and involves also the nucleus of the sixth nerve.

Deafness would point to a lesion at the base of the brain (pons), or at the internal auditory meatus.

Paralysis of the soft palate is suggestive of injury to the nerve within the aqueduct of Fallopius, and involving the great petrosal nerve to Meckel's ganglion.

Loss of taste and dryness of the mouth show that the lesion is probably within the canal before the chorda tympani is given off.

If both facial nerves are affected (rare), the lesion must be in the pons ; or there must be disease extending to both internal auditory foramina ; or there must have been peripheral injury to both nerves.

Prognosis should always be guarded if the cause cannot be ascertained. If the injury be situated externally to the stylo-mastoid foramen, recovery is probable. The less alteration there is in the electrical reactions of the palsied muscles, and the less chronic the affection, the better is our prognosis.

Treatment depends greatly on the cause. If there be a syphilitic history give iodide of potassium a prolonged trial. This drug is also useful in gouty and rheumatic subjects, or where there is reason to suspect neuritis in the aqueduct. We should also resort to the other usual methods laid down when discussing gout and rheumatism.

Counter-irritants (blisters, rubefacient liniments) should be applied to the face over the course of the nerve. If no relief be obtained, resort quickly to electricity. Apply faradic (interrupted) current to the muscles if they respond ; or, failing this, apply the galvanic current, slowly interrupted.

In weakly anæmic subjects, iron in combination with strychnia is of great service.

The Eighth, or Auditory Nerve,

arises by two roots which embrace the restiform body, viz. : a lateral root, which is distributed to the cochlea, and which is the true nerve of hearing ; and an anterior root, which is distributed to the vestibule, and which is concerned in maintaining equilibrium.

Its deep origin is from four scattered nuclei in the floor of the fourth ventricle. Its cortical centre is in the first temporo-sphenoidal convolution ; but as the centres are bilaterally associated, a destructive lesion of both sides is necessary to produce entire deafness.

Lesions may occur within the brain at its cortical centre, or between it and the pons ; or between its superficial origin at the postero-lateral border of the pons ; or at its periphery.

A central or intracranial lesion is rare. It produces 'word-deafness,' or inability to understand words, which although heard, are as a foreign language.

A nuclear lesion is equally rare.

A basic lesion may result from hæmorrhage, fracture of skull, tumour, and meningitis. Occurring in early life, the lesion would cause deaf-mutism.

Peripheral or labyrinthine lesions chiefly result from extension of disease from the middle ear. They produce auditory hyperæsthesia and tinnitus ; or diminished auditory sensibility (deafness) ; or vertigo.

In auditory hyperæsthesia the slightest sound occasions distress. Deafness, varying in degree from slight dulness of the auditory function to complete loss of hearing, is a common sequel to peripheral disease. This form of nerve deafness must not be confused with that variety which is due to obstruction to the waves of sound.

If the vibrations of a tuning-fork cannot be recognised when the instrument is away from the ear, but are heard when the handle of the fork rests on the temporal bone, the inference is that conduction is bad, but the nerve apparatus healthy.

TINNITUS AURIUM is the term used to denote distressing noises or roarings, pulsations, whirrings, and various other subjective sensations. It may occur from any form of ear disease. It is due to nerve-irritation, but especially of the periphery. Amongst the most frequent causes are, collections of cerumen irritating the membrana tympani, anæmia, and gout. It also not infrequently occurs in the premonitory stages of epilepsy and megrim; and also in hysteria and hypochondriasis.

Treatment.—The first indication is to attend to, and if possible remove, the cause. Anæmic and gouty patients must be treated according to the rules described under those diseases. Accumulations in the meatus must be removed by the syringe. As regards idiopathic cases, numerous drugs have been prescribed, and in many instances they fail to give relief. Gowers recommends salicylate of soda, and, in addition, quinine; iodides and bromides may be tried. Nitro-glycerine is often of service when tinnitus is due to arterial tension. Hydrobromic acid may also be prescribed.

AUDITORY VERTIGO (*Menière's Disease*). In this condition there is giddiness and staggering, often accompanied by buzzings in the ears and other signs of tinnitus, together with symptoms which somewhat resemble epilepsy (*petit mal*). The patient is suddenly attacked with noises in the ear, accompanied by reeling or staggering; or the surrounding objects appear to be turning round him. Occasionally there is slight and temporary insensibility. The attack passes off in a few minutes, leaving the patient pallid and faint, with a clammy skin. Occasionally vomiting supervenes on recovery.

Other principal features of the disease are the paroxysmal nature of the attacks, the constant direction in the apparent movements of surrounding objects, and in the staggering movements of the patient himself.

On the supposition that the disease is one which involves the centres of hearing and of equilibrium, it is probable that the tinnitus and vertigo are of the nature of discharge of energy from these centres; and hence the staggering movements may be forwards or horizontal, according to the semi-circular canal which is principally involved, whether vertical

or horizontal. In support of this are the facts: (1) that the movements of the patient himself and of surrounding objects are towards the affected side; (2) experimental research has shown that lesions of semicircular canals cause disturbances in equilibrium, the vertigo varying in its direction according to the canal which is injured.

Treatment.—In view of the possible relation of Menière's disease to epilepsy, bromide of potassium or ammonium should be given freely (gr. xv. t.d.). Salicylic acid and gelsemium are also valuable remedies. Blisters, and counter-irritation by iodine, may be applied behind the ears.

The rules of diet and general health apply equally well to this disease as to epilepsy.

The Ninth, or Glosso-pharyngeal Nerve,

passes through the middle division of the jugular foramen, in a separate compartment of dura mater, in front of and internal to the tenth and eleventh nerves. Its deep origin is from a nucleus in the floor of the fourth ventricle below and behind the auditory nerve. The nuclei of this nerve and of the vagus and spinal accessory are really inseparably connected.

The primary function of this nerve is probably motor only to the middle constrictor and to the stylo-pharyngeus muscles; and if it has taste fibres they probably come to it from the fifth through the otic ganglion.

Owing to lesions which affect the nucleus of the vagus and other nerves, in common with the glosso-pharyngeal, it is difficult to differentiate which symptoms are due to injury to this latter nerve. Anyway, nuclear lesions of the glosso-pharyngeal are rare; but the trunk may be involved in basic meningitis and cerebral tumour.

On the supposition that it does possess the function of taste, we may try this function by acids, bitters, and sweets. The electric current applied to the tongue also produces a characteristic metallic taste. It is important to select such tests as have no odour, as much of the function of taste depends on the odour of the substance. Vinegar, quinine, and sugar are the usual tests which are used.

The Tenth, or Vagus Nerve,

arises from a nucleus in the floor of the fourth ventricle, external to that of the twelfth. Its cortical centre is unknown.

The nerve traverses the middle division of the jugular foramen in the same compartment as the spinal accessory. It consists of sensory and motor fibres, and is distributed to the pharynx, œsophagus, and stomach, and also to the larynx, lungs, and heart. It is a nerve of special function principally, its motor fibres probably being communicated by the spinal accessory.

The nerve may be implicated by disease at its nucleus, at its superficial origin, in its course, or at its peripheral distribution.

Nuclear lesions also usually involve, at the same time, the glosso-pharyngeal, spinal accessory, and hypoglossal nerves, as in bulbar paralysis.

The nerve may be compressed at its superficial origin by tumour or by basic meningitis.

In its course down the neck it may be the subject of surgical or other injuries, or it may be compressed by a goitre or other tumour.

The peripheral distribution is very extensive, and consequently the affections of lesions are complex and varied. The following classification, adapted from Osler, is perhaps the most convenient.

(a) *Pharyngeal Branches*.—Irritation of these nerves gives rise to difficulty of deglutition, and often causes the food to pass into the windpipe. General spasm of the pharynx occurs in hydrophobia and occasionally in hysteria.

(b) *Gastric and Œsophageal Branches*.—Lesions of these nerves cause spasm of the gullet, vomiting, gastralgia, and other symptoms when the irritation is peripheral. The gastric crises of locomotor ataxia are caused by central stimulation.

(c) *Laryngeal Branches* supply the muscles of phonation. The recurrent nerves contain fibres of distribution both to the abductor muscles and to their opponents the adductor muscles.

But in nearly all paralytic affections of the recurrent nerves, the abductors are the first to fail, so that the cord or cords are fixed near the middle line. This is quite in keeping with the view expressed elsewhere (pp. 484, 717, 768), that throughout the body the flexors and adductors are collectively stronger than the extensors and abductors. The most important morbid conditions related with these nerves are abductor and adductor paralyses, which may be unilateral or bilateral.

- (i) *Unilateral Abductor Paralysis* is due to pressure on one recurrent nerve. Thoracic tumour, especially aortic aneurysm, is the most frequent cause, the left posterior crico-arytenoid muscle therefore is paralysed. It occasionally occurs on the right side, from malignant growth in the œsophagus pressing upon the right recurrent nerve.

The voice is harsh and hoarse, and the affected cord does not move in inspiration; but dyspnœa is not a marked symptom.

- (ii) *Bilateral Abductor Paralysis* is due to palsy of both posterior crico-arytenoid muscles. It may be caused by laryngeal catarrh, by pressure on both vagi trunks, or on both recurrent laryngeal nerves; or it may be due to a central lesion, as in locomotor ataxia and bulbar paralysis.

The chief symptoms are inspiratory whistling stridor, urgent dyspnœa, and loss of voice. The cords are seen to be motionless and closely approximated. In deep inspiration they may even touch each other.

- (iii) *Adductor Paralysis* is caused by palsy of the arytenoid and crico-aryteno-lateralis muscles. It occurs in hysteria and in laryngeal catarrh. The patient is aphonic, and on laryngoscopic examination the glottis is found normally open, but the cords fail to come together during the attempt at phonation.

(d) *Pulmonic Branches*.—Some supply the muscular coats of the bronchi; others convey impulses up to the respiratory centre. The spasm of asthma is probably due to a peripheral irritation.

Paralysis or exhaustion of the medullary centre gives rise to an alteration in the respiratory rhythm, known as *Cheyne-Stokes Respiration*. This phenomenon 'consists in the occurrence of a series of inspirations increasing to a maximum, and then declining in force and length until a state of apnoea is established. In this condition the patient may remain for such a length of time as to appear to be dead, when a low inspiration, followed by one more decided, marks the commencement of a new ascending and then descending series of inspirations' (Stokes).

(e) *Cardiac Branches*.—Irritation of the cardiac branches of the pneumogastric causes slowing in the heart's action. The stimulus may be supplied in the thorax, or in the neck, by the pressure of a tumour. Compression of the carotids during a surgical operation may produce a similar effect.

Extreme slowness of the heart (Bradycardia) occurs in bulbar disease and in epilepsy. But it may also occur in perfectly healthy and athletic men.

Rapid action of the heart (Tachycardia), on the other hand, occurs in paralysis of the vagi.

There are also some sensory fibres in the pneumogastric, by which the distress of palpitation and other cardiac disturbances are conveyed to the sensorium.

The Eleventh, or Spinal Accessory Nerve,

consists of a spinal or motor portion, which arises from the motor cells of the cord as low down as the sixth cervical nerve, and a bulbar or accessory portion, which rises by four or five fasciculi from the medulla in conjunction with the vagus. The spinal portion emerges between the posterior nerve-roots and the ligamentum denticulatum, then enters the skull by the foramen magnum, where it joins the accessory part. The compound nerve thus formed passes out through the middle compartment of the jugular foramen.

The part which is accessory to the vagus supplies the muscles of phonation; the other, or spinal portion, supplies the sternomastoid and that part of the trapezius which is between the occiput and the acromion of the scapula. Consequently the nucleus

of the accessory portion may be involved in medullary lesions ; the nuclei of the spinal portion may be implicated in degeneration of the anterior cornua of the cervical grey matter.

The main trunk is liable to pressure from meningitis or tumours.

The symptoms of disease of the accessory part are those of laryngeal palsies (see p. 666).

Disease of the cervical portion causes paralysis of the sterno-mastoid and trapezius of the same side. If the nerve be impinged upon between its exit from the jugular foramen and its piercing the sterno-mastoid muscle, both this muscle and the trapezius would be affected. The trapezius alone may be palsied by lesion of the nerve during its passage through the posterior triangle of the neck.

When the symptoms are complete, the patient can only rotate the head to the opposite side with difficulty ; the fold of the neck is flattened or hollowed ; the shoulder drops ; the patient cannot shrug ; the lower angle of the scapula is rotated towards the spinal column by the unopposed action of the levator anguli and the rhomboids ; and the arm cannot be raised above the head, since the scapula is no longer fixed for the deltoid to act upon.

Atrophy and degenerative changes occur in the paralysed muscles if the nerve injury is permanent.

The Twelfth, or Hypoglossal Nerve,

arises from a long nucleus in the lower third of the floor of the medulla, close to the middle line. This nucleus is continuous with the anterior cornual cells of the cord.

The cortical centre is in the lower extremity of the ascending frontal convolution, immediately behind the vertical limb of the Sylvian fissure.

The nerve pierces the dura mater in two bundles, which unite as they traverse the anterior condyloid foramen. This nerve supplies directly, or indirectly by its descending branch, almost all the muscles which are attached to the hyoid bone.

A lesion above the nucleus causes palsy of the tongue on the opposite side, and hemiplegia ; but the paralysed muscles

of the tongue neither waste nor lose their electrical reactions.

A nuclear lesion may occur in bulbar paralysis. Usually the lesion and its effects are symmetrical, since the nuclei are closely approximated to each other.

The superficial root and trunk of the nerve may be involved in meningitis, tumour of the base of the brain, or in disease of the occipital bone.

The tongue is paralysed on the side opposite to the lesion in cortical disease ; but on the same side as the lesion in medullary disease. When the organ is protruded, it is pushed over to the palsied side by the healthy muscles of the opposite side ; and it may present fibrillar movements. Speech is not much affected.

Atrophy of the tongue ensues on nuclear disease. It is specially shown in the mucous membrane, which becomes wrinkled.

In bilateral (nuclear) disease the tongue is symmetrically atrophied, and lies motionless on the floor of the mouth. Speech and mastication are necessarily impaired.

DISEASES OF THE SPINAL CORD

ANATOMY

The spinal cord is from fifteen to eighteen inches long. It extends from the margin of the foramen magnum above, to the lower part of the first lumbar vertebra below. At birth it extended to the third lumbar vertebra; but the bones growing faster than the cord, it appears in the adult as though the cord had been drawn upwards, with the result that the nerves arise from the cord, not opposite their intervertebral foramina, but at points varying from one to three vertebræ higher up.

The shape of the cord is cylindrical, but slightly flattened from before backwards. It presents two enlargements: a cervical bulb which extends from the third cervical to the first dorsal vertebra, and a lumbar bulb which extends from the tenth dorsal vertebra to the end of the cord, being largest opposite the last dorsal vertebra. These enlargements, which are absent in the fœtus, develop with the growth of the limbs.

The cord is invested by the vascular pia mater, which extends into its anterior fissure; and is further enveloped by the dura mater, the space between the two coats being regarded as a cavity lined by serous membrane, or arachnoid.

The dura mater differs considerably from that of the brain. Thus, it does not contain venous sinuses; it does not act as periosteum to the vertebræ; it is less firmly attached to bone; and between the membrane and the bony neural canal is a well-marked space which contains areolar tissue, fat, blood-vessels, and lymphatics.

In transverse sections of the cord it will be seen that the

superficial parts of the cord are composed of white matter, which consists for the most part of nerve fibres ($\frac{1}{150}$ to $\frac{1}{1500}$ of an inch) which run longitudinally, whilst the interior of the cord is composed of grey matter which is especially developed in the cervical and lumbar bulbs. The nerve-fibres are held together by a fine connective tissue or neuroglia, which also contains many granular bodies or modified connective-tissue cells.

Two obvious fissures are also seen, the anterior and posterior median, which divide the cord into two symmetrical halves, connected by a white and a grey commissure at the bottom of the anterior and posterior fissure respectively.

Secondary fissures formed by the lines of exit and of entry of the anterior and the posterior nerve roots, divide each half of the cord into three primary columns, viz. the anterior, the lateral, and the posterior.

The anterior column is again subdivided into an internal column of Türck, or the direct pyramidal tract, and a column which is between this and the anterior nerve roots.

Similarly the posterior column is subdivided into an internal fasciculus of Goll, and an external column of Burdach or posterior root-zone. In the posterior median column of Goll the connective tissue is most abundant. This is important, as in some instances a normal histological appearance has been mistaken for a pathological change.

The lateral column also consists of at least four distinct bundles of fibres, viz. : (i) the **CROSSED PYRAMIDAL TRACT** ; (ii) the **DIRECT CEREBELLAR TRACT** ; (iii) the **ANTERO-LATERAL ASCENDING TRACT** ; and (iv) a **SMALL TRACT**, the function of which is unknown, and which skirts the outer surface of the grey matter between the horns.

(i) The **CROSSED PYRAMIDAL TRACT**, the most important, is situated between the grey matter and the direct cerebellar tract. It contains nearly all the fibres from the opposite anterior medullary pyramid. It is a large bundle in the upper parts of the cord, but dwindles as it reaches the lumbar region, where it becomes superficial, and is found immediately external to the posterior horn.

(ii) The **DIRECT CEREBELLAR TRACT** is situated on the lateral surface, and therefore external to the preceding. It conducts impressions to the cerebellum.

(iii) The **ASCENDING (ANTERO-LATERAL) TRACT** is situated immediately in front of the crossed pyramidal tract, and conducts impressions upwards probably to the cerebellum.

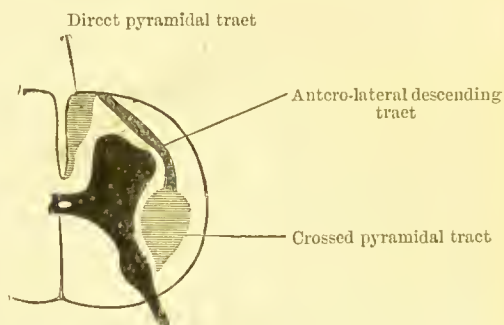


FIG. 31.—**DIAGRAM OF DESCENDING DEGENERATIONS.**
(Lesion below decussation)

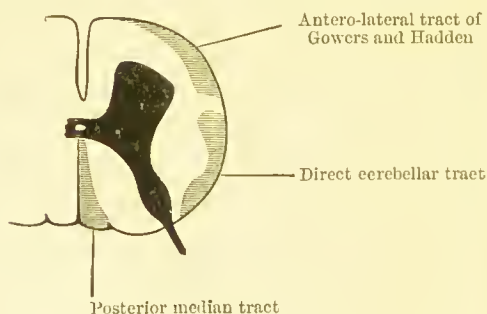


FIG. 32.—**DIAGRAM OF ASCENDING DEGENERATIONS**

(iv) The **FOURTH FASCICULUS** is immediately external to the outer surface of the grey matter, being limited externally by the crossed pyramidal tract. As stated, its function is not definitely known, but it may possibly be commissural.

The direct pyramidal tracts contain those motor fibres from

the brain which have not decussated at the medulla ; they are not found below the mid-dorsal region. In the posterior columns, those fibres which constitute the posterior median fasciculus of Goll are in direct continuity with the posterior nerve roots and undergo degeneration after destructive lesion of these nerve roots ; whilst the postero-external fasciculus contains such fibres of the posterior nerve roots as do not immediately enter the grey matter. After entering the grey matter they decussate across the middle line immediately, except in the lower third of the cord ; here they run up some little distance before crossing over.

It will thus be seen that in each half of a given segment of the cord *descending* lesions, the result of injury to the motor centres of the brain, may manifest themselves by Wallerian degeneration in two distinct tracts, viz. the crossed and the direct pyramidal. According to Sherrington, there is a third antero-lateral tract which is situated between the surface of the cord and the anterior horn. It is, however, so diffuse, and mixed up with others 'that it hardly deserves to be called a tract' (see fig. 31).

Similarly, *ascending* lesions from injury to the cord are shown in three tracts, viz. : the posterior median, the direct cerebellar, and the ascending antero-lateral tract of Gowers and Hadden (see fig. 32).

The grey matter is arranged in two crescent-shaped masses joined by a transverse commissure. Each crescent has two well-marked horns, anterior and posterior ; the posterior is long and narrow, and comes to the surface at the posterior lateral fissure ; the anterior is rounded ; it does not come to the surface, only extending to the rootlets of the anterior nerve roots. The outer side of each crescent is rugged in appearance, enclosing a few tubules of the white matter. The grey commissure is situated at the bottom of the posterior median fissure ; it contains a small canal or ventricle, which extends from the calamus scriptorius above, to the lower extremity of the cord.

In the grey substance the fibres are half the diameter of those in the white portions. They constitute a complete

interlacing network, embedded in which are found the large multipolar cells, each cell containing a nucleus and a bright nucleolus. These cells are for the most part arranged in three groups, viz. : (i) in the anterior horn ; (ii) in the middle and towards the inner aspect (posterior vesicular column) ; and (iii) also in the middle, but towards the outer surface of the grey matter (tractus intermedio-lateralis).

The function of the grey matter, so far as our knowledge goes, is to afford a presiding trophic influence over the skin and viscera. The group of cells in the anterior horns is concerned in reflex actions and in the nutrition of joints ; they also act as trophic centres for muscles and their motor nerves.

Thus the cord, as a whole, is the great channel by which motor and sensory impulses are conveyed from and to the brain ; it contains the centres for many important reflex acts, some of which are controlled by the higher centres in the brain, others again which are without such control ; it is also the seat of trophic centres for the muscles, bones, and joints of the trunk and limbs, and for the viscera. Besides, it has the centres for the sexual organs, bladder and rectum, and the vaso-motor actions.

It is to be regarded not so much, therefore, as an elongated mass of nerve tissue, as a chain, each segment or link of which acts not only as a conductor of impulses, but also as a centre by itself with definite functions, with afferent and efferent nerves in connection.

An admirable succinct description of the cord and its functions is found in Suckling's 'Diagnosis of Diseases of the Nervous System.'

The following table, copied from Gowers' 'Diseases of the Spinal Cord,' will also be found useful to the student. It shows 'the approximate relation to the spinal nerves of the various motor, sensory, and reflex functions of the spinal cord,' and will be found of great value in localising transverse lesions at different levels.

Motor		Sensory	Reflex
	C 1) Small-rotators of head	1)	1
St. mastoid	2) Depressors of hyoid	2) Scalp	2
Upper neck muscles	3) Levator ang. scapulae	3) Neck and upper part of chest	3
Upper part of trapezius	4) { Diaphragm {	4) { Shoulder	4
	5) { Serratus { Shoulder muscles	5) { Arm, outer side	5
	6) { Flex. of elbow {	6) { Radial side, forearm and hand; thumb	6
	7) { Supinators {	7) {	7
Lower neck muscles	7) Ext. wrist and fingers		
	8) { Ext. elbow {		
Middle part of trapezius	8) { Flex. wrist and fingers {	8) { Arm; inner side; ulnar side of forearm and hand; tips of fingers	8
	8) Promotors		
	8) Muscles of hand		
	D 1)	1)	1
	2)	2)	2
	3)	3)	3
Lower part of trapezius and dorsal muscles	4)	4)	4
	5) Intercostals	5) Front of thorax	5
	6)	6) Eusiform area	6
	7)	7)	7
	8)	8)	8
	9)	9)	9
	10) Abdominal muscles	10) Abdomen (Umbilicus 10th)	10
	11)	11) Buttock, upper part	11
	12)	12)	12
	L 1)	1)	1
		1) Groin and scrotum, front	1
Lumbar muscles	2) Cremaster	2)	2
	3) Flexors of hip	3)	3
Peroneus longus	4) Extensors of knee	4) Thigh { outer side	4
Flexor of ankle	4) Adductors of hip	4) { front	
Ext. of ankle	4) Ext. and adduct. of hip	4) { inner side	
	5) Flexors of knee	5)	5
	S 1)	1) Leg, inner side	1
	1) Intrinsic muscles of foot	1) Buttock, lower part	
	2)	2) Back of thigh	
	3) Perineal and anal muscles	3) Leg and { except foot { inner part	3
	4)	4) Perineum and anus	4
	5)	5) Skin from coccyx to anus	5
Co		Co	Co

SPINAL MENINGITIS

Definition.—Inflammation of the membranes of the spinal cord.

Causation.—It may result from the effects of blows, or falls, or of various injuries to the spine. It may also be secondary to the influence of new growth, cold, or to some

constitutional disease, such as rheumatism, gout, syphilis, or to some specific fever. The majority of cases, however, appear to be secondary to injury or disease of the vertebral column, such as fractures or caries. Occasionally it may be secondary to an acutely spreading bed sore. Lastly, it may occur as a primary disease, without any known cause, other than that the subjects are weakly, and liable to any form of inflammation.

Pathology.—Two varieties are usually described, which are based entirely on anatomical relations. Thus, that form in which the inflammation begins internally to the dura-mater sheath is spoken of as *m. interna*; whilst that which commences external to the dura mater, involving it and extending to, or indeed originating in the packing of fat and areolar tissue between it and the vertebrae, is known as *m. externa* or pachymeningitis.

The internal variety may be diffuse, extending over a large area of spinal cord, and probably always extends to the dura mater if the patient survive any length of time.

Meningitis externa, on the other hand, whilst it may be diffuse, may also be limited to a comparatively small area, the inflammation apparently spending its force in the peridural space.

Whichever form of meningitis occurs, the pathological processes are much the same. Three stages are usually described.

The *first* stage is characterised by marked hyperæmia of the membranes. The pia mater especially shows this. It is red, its capillaries injected, and here and there presents punctiform ecchymoses, or more extensive extravasations. The arachnoid shows a similar change, but in a less obvious degree. The congestion also at times extends to the capillaries which ramify from its surface towards the interior. In the *second* stage there is generally a sero-purulent exudation, with fibrinous deposit on the membranes and cord. The pia mater and arachnoid consequently become thickened and opaque; the surface of the cord is somewhat rough and sticky, from fibrinous exudation, this being most pronounced at its posterior surface. The cord itself on section is seen to be

oedematous and somewhat paler than normal (meningo-myelitis).

The *third* stage terminates either in absorption, or in adhesion with thickening of the membranes, and chronic inflammatory changes of the surface of the cord (sclerosis). If absorption occurs, the vessels become less turgid, the sero-purulent fluid dries up, leaving the membranes here and there thickened, and perhaps adherent.

When the inflammation is limited to the dura mater and parts external to it, the inflammatory products, usually purulent, extend to a variable distance up and down the vertebral canal. In caries it is not infrequently limited to the immediate locality of the affected vertebræ. The inflammation causes absorption of the fatty and areolar tissues surrounding the cord; and usually pus in greater or smaller quantities forms in this space. Occasionally the congestive process extends inwards to the pia mater; but more usually causes annular thickenings of cicatricial tissue, which may cause compression of the cord and subsequent ascending and descending degenerations, or give rise to permanent adhesions to the vertebræ.

Symptoms.—In the internal form of meningitis the symptoms are usually sudden in their onset and well marked. The initial stage is characterised by well-marked rigors, a quick pulse, and high fever. These are succeeded by symptoms pointing to irritation or compression of the nerve roots. Thus we find pains, severe and deep in the back, extending down the spinal column, and radiating to the trunk generally, and limbs. These pains are intensified by the slightest movements of limbs or body, and are accompanied by twitchings or rigid spasms of the muscles of the neck and back, and possibly also of the extremities, due to irritation of the anterior nerve roots and lateral columns of the cord.

Subsequently, from compression of the nerve roots by the serous effusion, paralysis ensues, both motor and sensory, and loss of reflexes. Exaggeration or other modifications of these symptoms will depend on the locality of the inflammation if it be a limited one. Thus when the cervical region

is affected as far up as the medulla, we should expect to find stiffness of the muscles of the neck, with retraction of the head, difficulty in swallowing, an irregular pulse, embarrassed breathing, and pains, with spasmodic movements, in the muscles of the arms. If the dorsal region is involved, it would give rise to pains extending to the abdominal walls and to the lower limbs, stiffness in the muscles of the back, and tension of the muscular belly walls. Affection of the lumbar region would be principally indicated by paralysis of the bladder and rectum, with hyperæsthesia over the hypogastric region.

The symptoms of *m. externa* are much the same. There are severe pains in the back and limbs, increased on movement. There is tenderness over the spinous processes, especially at the area of lesion, if it be a local one. This is soon followed by hyperæsthesia, and possibly by anæsthesia, paralysis of muscles, the rapid formation of bedsores, and other symptoms which point to irritation and injury of nerve roots.

The formation of pus may cause symptoms of compression of the surface of the cord, viz. flaccidity and palsy of muscles, loss of all reflexes, together with profuse sweating, rigors, and an extremely high temperature (105° to 107°), as in pyæmia.

Prognosis.—Generally unfavourable. Comparatively few recover. In the severe acute type, death occurs in a few days, usually from asphyxia. It may, however, run a chronic course and prove fatal by asthenia.

Diagnosis.—(1) FROM MYELITIS.—In this disease the early and complete motor paralysis is the chief symptom. There is no spasm or rigidity of the dorsal muscles; the febrile symptoms are less pronounced; nor is there such marked pain in the back extending to the limbs.

(2) FROM TETANUS.—Here the muscular spasms are more violent, sudden, and general; they also involve the masseters, and there is trismus. Intermissions of spasm are common; but hyperæsthesia and anæsthesia, in any marked degree, are absent.

Treatment.—Perfect rest in bed; the patient should lie in such position as is found to be attended by least pain and discomfort. The bowels should be purged. For this, mercury is the best drug, as it appears at the same time to check the

inflammatory progress. Give it in repeated small doses until the gums become soft. Gowers recommends the oleate of mercury rubbed along the spine. In the early stages, dry cupping, leechings, or blisterings to the spine should be ordered. Hot baths and packings have also been recommended, but we cannot advocate this treatment in the face of the exquisite torture caused by movements. Morphia or opiates are not contra-indicated; on the other hand, they afford immense relief, and procure sleep. Iodides may be given when the acute symptoms have passed away. But their influence in removing the products of inflammation is problematical.

The further points to be remembered are the avoidance (if possible) of bedsores, and regular attention to the urinary bladder.

MYELITIS

Definition.—Inflammation of the spinal marrow. The term is generally restricted to the acute type.

Causation.—Often obscure. (1) It may supervene as the result of direct *exposure to cold* or to *heat*; after (2) *severe exertion*; (3) from the effects of *injuries to the cord*; or (4) from *inflammatory extensions*, secondary to carious vertebrae or to spinal meningitis. (5) It may be a sequel of *specific fevers*, such as typhoid, diphtheria.

Pathology.—The inflammation may only extend to a certain depth in the cord, or it may be limited to a complete narrow, transverse lesion; or horizontally to one half of the cord; or, finally, it may be diffuse, involving the cord to a more or less considerable vertical extent.

It may commence in and be limited to the superficial surfaces of the cord, when secondary to meningitis, or it may commence in the grey matter. The latter is more frequent in idiopathic myelitis.

The microscopical changes are difficult to follow beyond the congestive and exudative stages. The capillaries are engorged; serous exudation takes place in the perivascular spaces, followed by migration of blood corpuscles both red and white. The cord at this stage is somewhat red on section.

The subsequent result of the inflammatory changes is seen in the acute softening which takes place. At the part inflamed the marrow becomes pulpy, or even creamy, in consistence ; it easily breaks down under a stream of water ; large hæmorrhages occur as a result of the want of support to the vessels, and the cord is thus completely disorganised. The formation of pus is rare, although it may occur. It is much more frequent in encephalitis, but in myelitis it is probable that the patient dies too soon for pus formation to take place.

Symptoms.—The onset of the disease may be terribly sudden (acute), or it may supervene gradually (chronic). The typical symptoms are most marked in an acute case of transverse myelitis, and are such as would accord with our knowledge of the various functions of the cord. Thus the patient complains of general malaise ; tenderness of vertebræ over the site of the lesion ; pain in the back, or girdle pains, with a tingling or numbness or a sense of cold in the limbs, together with twitchings or convulsions in the muscles. There is at the same time a slight febrility (100° to 102°). Then, in a few hours, there is motor and sensory paralysis of the legs and all those parts whose nervous supply is from the cord below the seat of inflammation.

(*Motor*) All the muscles which are supplied by the nerve cells of the anterior cornua at the seat of lesion, and all muscles supplied by nerves below this, are paralysed. Those muscles supplied from the affected area of the cord rapidly waste and lose their faradic contractility ; but wasting and altered electrical reactions do not occur in those muscles supplied from below. The patient has loss of control over the sphincters ; and the urine becomes alkaline.

If, however, the lesion is in the lumbar region, the urine becomes decomposed from cystitis ; if the lesion be above the lumbar region, the reflex defæcatory acts take place entirely without control or knowledge.

(*Sensory*) Paralysis of sensation is early and complete below the lesion, and in the area supplied by the inflamed segment. At the upper level of the lesion there is hyperæsthesia, due to irritation of the sensory nerves here ; thus hyperæsthesia extends

to the arms in cervical myelitis, to the legs in lumbar myelitis, whilst in dorsal myelitis there is a band of hyperæsthesia round the trunk which would, as it were, represent the position of dorsal limbs. Further, bedsores rapidly form, owing to the mechanical pressure on parts thinly covered by skin and muscle, and also owing to trophic changes in those parts supplied from the inflamed cord : in other words, a peripheral lesion. The reflexes also give valuable evidence. Those above the lesion are normal, those springing from the level of the lesion are lost, whilst those below are increased. The parts which are paralysed are warmer than normal, owing to vaso-motor paralysis. Death occurs usually in a few days ; or it may be postponed for some weeks or months, when the patient succumbs from exhaustion. Previously to this, however, degeneration of the pyramidal tracts below the lesion has occurred, as evidenced by rigidities and contractions. The above description will apply to the ordinary form of transverse myelitis ; but the extent and severity of the symptoms will, of course, vary according to the locality and intensity of the inflammation.

Prognosis.—Nearly always unfavourable. Considerably influenced by (1) *Region of Lesion*.—If in the cervical region, it is of bad omen. If in the dorsal region, the outlook is fairly favourable ; whilst in the lumbar region the prognosis is again gloomy, but not so bad as if in the cervical cord. (2) *Lateral Extent of Lesion*.—When the grey matter is involved, the prognosis is unfavourable, owing to the appearance of bedsores and other trophic changes. (3) *Vertical Extent of Lesion*.—Obviously the greater length of cord affected, the more severe the symptoms and the greater the danger. Exception must be made in the cases of cervical myelitis due to caries. Cases not unfrequently recover after ankylosis of vertebrae has taken place.

Diagnosis.—FROM SPINAL MENINGITIS.—In this latter disease intense shooting pains in the course of the compressed nerves, together with muscular spasms, which increase the pains, are the most pronounced symptoms. The muscles also are not so profoundly paralysed ; and absolute anæsthesia is, perhaps, never found. As to the probable cause of the myelitis,

it may be observed that, as a rule, in the idiopathic form, resulting from colds and fevers, loss of sensation is the earliest and most marked symptom (grey matter) ; whilst in those cases which are secondary to extension from bone or meninges, loss of motion precedes that of loss of sensation.

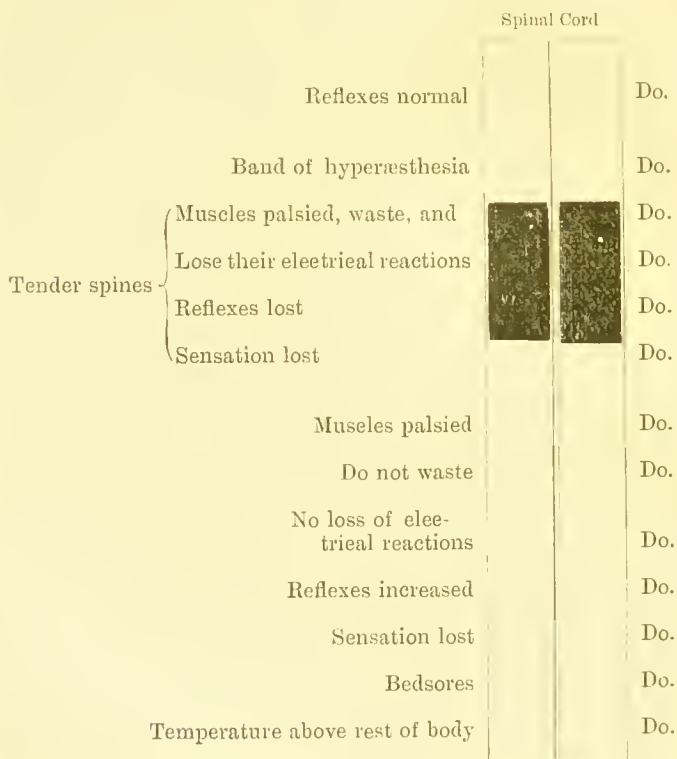


FIG. 33.—DIAGRAM TO ILLUSTRATE SYMPTOMS IN TRANSVERSE MYELITIS.
THE DARKENED PORTION REPRESENTS THE SEAT OF LESION

Treatment.—The patient should be placed on a water-bed, and should, if possible, have the pelvis raised, or he should lie prone on his face. Scrupulous attention to cleanliness and other methods to avoid the formation of bedsores, are imperative. We may order ice-bags to the spinal column, or apply dry cupping, blisters, or even the actual cautery over the lesion,

with a view to arrest inflammation. On similar grounds, ergot and belladonna may be given so as to produce contraction of the vessels of the cord. Purgatives are necessary from time to time, and the bladder must be periodically relieved by catheter. Any tendency to cystitis should be obviated by repeated washings out of the bladder with weak acids or other suitable application. The exhibition, by the mouth, of benzoate of ammonia—which, by its decomposition, keeps the urine acid—is, however, preferable to the use of the catheter. In the later stages, if the patient survive so long, we may prescribe strychnia to counteract the anæmia of the cord. A generous dietary should be allowed, supplemented by iron tonics, cod-liver oil, or preparations of the phosphates.

Hemilateral Myelitis

Definition.—Inflammation of one half of the horizontal plane of the spinal cord.

Symptoms.—The symptoms are much the same as those attending complete myelitis, with certain modifications which are accounted for by the histological arrangements and the functions of the fibres in the cord. Thus—(1) (*Motor*) there is paralysis, on the same side as the lesion, of all muscles which are supplied by the cord, from the point of inflammation downwards; and those muscles which are supplied by the affected portion of the cord eventually atrophy and present qualitative electrical changes. (2) (*Sensory*) There is complete anæsthesia, on the opposite side, of all parts below the lesion; whilst on the same side there is hyperæsthesia. The cause of this latter phenomenon is as yet obscure; but the pain produced by even gently touching the skin on the same side is often most intense. In addition, there is a symmetrical band of anæsthesia extending round the body at the level of the lesion. This is easily understood when it is remembered that the sensory fibres run into the cord and decussate at different levels; and therefore a myelitis, however narrow, would arrest sensory impulses, not only from the opposite side, but also those conveyed by nerves of the same side which have not yet decussated. There is also a symmetrical band of hyperæsthesia,

immediately above the lesion, conveyed by the extension of inflammatory irritation to the sensory nerves there. The *reflexes* of the inflamed segment are lost; those which pass through the cord below it are increased. There is heat from vaso-motor paralysis on the same side as the lesion. (See diagram, modified after Gowers.)


Spinal Cord		
Reflexes normal		Do.
Band of hyperæsthesia		Do.
Band of anæsthesia		Band of anæsthesia
Reflexes lost		
Motor palsy		Motor power unaffected
Hyperæsthesia		Anæsthesia
Reflexes increased		Reflexes unaffected
Temperature above the rest of the body		Temperature same as the rest of the body

FIG. 34.—DIAGRAM TO ILLUSTRATE SYMPTOMS IN HEMI-LATERAL MYELITIS

Treatment.—As in the ordinary transverse form.

ACUTE ASCENDING PARALYSIS (LANDRY'S PARALYSIS)

Definition.—A progressive, acute paralysis, commencing in the lower extremities, thence extending to the trunk and upper limbs, and causing death by involving those parts and organs which are supplied by the medulla.

This disease does not appear to be an actual disorder of the cord, but it is considered here for convenience.

Causation.—*Age and Sex.*—Most frequent in early manhood, between twenty and forty years of age. Occasionally but rarely seen in women and children. It is met with during convalescence from *small-pox*, *typhoid*, *diphtheria*, or other *specific fevers*. It has, presumably, been found to be consequent on *exposure to cold*, *injuries* and *wounds*, or (4) the result of *alcoholism* and *syphilis*.

Pathology.—No definite lesion has been discovered in the nervous system. The brain and spinal cord may be quite normal and healthy. Occasionally neuritis has been discovered, but this pathological change is by no means constant. The probability of the disease being produced by some toxic agent is supported by the acute congestion of the spleen and lymph-glands which is found after death. The organisms which may have produced this toxæmia have not been discovered ; and the assumption is that the system has by some means got rid of these organisms, although the poisonous chemical effects produced by them have remained.

Symptoms.—The disease is essentially an acute one. It comes abruptly and runs a rapid course. The first symptoms complained of are a general malaise, with tingling or numbness in the extremities. Then paralysis supervenes in the lower extremities. The legs, beginning with muscular weakness, soon lose all motor power entirely, and in a few hours they lie completely motionless, helpless, and limp. Subsequently a similar condition invades the muscles of the loins, pelvis, abdomen, and trunk generally ; and thence extends, in order, to the muscles of the upper limbs and of the neck, to the diaphragm, and to the muscles which are supplied by the medullary nerves (lips, jaws, &c.). Occasionally however, in its upward course, some muscles appear to be spared by the disease ; but whenever the paralysis affects a part, the paralysis is complete and absolute. The muscles, however, do not waste ; there is no ‘reaction of degeneration ;’ and there are no rigidities, and no increase in the deep reflexes ; indeed, the latter phenomena are often lost. The patient does not lose control over the bladder or rectum, he does not lose cutaneous sensibility, and he retains his intellect unimpaired throughout, although unable to express himself either by speech or gesture.

Occasionally there can be detected, during the course of the disease, an enlargement of the spleen and of the mesenteric glands, as in other acute diseases. But febrility in this disease is seldom a marked feature.

Although not resembling it in all its characteristic symptoms, Landry’s paralysis bears, in many of its features, a

strong similarity to the paralytic form of rabies seen in some of the lower animals.

Diagnosis.—FROM ACUTE MYELITIS.—This condition gives rise to impairment or total loss of sensation, and the rapid formation of bedsores, symptoms which are absent in Landry's paralysis.

FROM MULTIPLE NEURITIS, see p. 720.

Prognosis.—Extremely unfavourable. Death occurs in from forty-eight hours to a few days. The patient rarely survives a week. Still, cases do recover; the progressive course of the paralysis becomes arrested, and the muscles gradually recover their lost power, the arms and those parts last involved being the first to show amelioration.

Treatment.—As we are yet in the dark as to its true pathology, treatment can only be empirical. We may, on general grounds, order counter-irritation to the spine, either in the form of constant sinapisms, or the actual cautery; hot baths also may be given. Gowers records a case which recovered after ergotin had been given every hour. In view of the toxæmia theory, tincture of the perchloride of iron in large doses (℥xxx.) is indicated. If there is any indication of syphilis, iodides with mercury should be given in large doses. The mercurial preparation had best be injected subcutaneously, so as to obtain its rapid action. Usually, however, there is no time for this, as the case runs a course which is too acute.

ACUTE ANTERIOR POLIO-MYELITIS (INFANTILE PARALYSIS)

Definition.—A paralysis supervening suddenly in young children, with fever, and terminating with paralysis and atrophy of certain groups of muscles.

Causation.—*Age.*—Before the tenth year; most cases observed in the second year, or during the period of eruption of the milk teeth. *Febrility.*—It is often secondary to some specific fever; or it may supervene on any febrility, such as that attending dentition and catarrhs of all forms. It is possible also that it commences with a feverishness which

is peculiarly its own. *Dentition* appears to be a strong exciting cause, but whether from nerve irritation which it causes, or from the fever which often accompanies it, is not quite clear. *Season of Year*.—Most cases have been observed during the hot months of summer; nevertheless, the influence of *cold* and *damp* has in many instances been thought to be an exciting factor, notwithstanding prevalence of the disease during summer.

Pathology.—The lesion occurs in the anterior horn of grey matter. In the acute stage the appearances would be those of an acute myelitis limited to this part. The vessels are engorged, extravasation of blood occurs, and the multipolar cells, and also the neuroglia, are swollen.

In chronic cases, from which most investigations have been made, the effects of a chronic local myelitis are seen. The side of the cord is shrunken and hard, and the motor nerve roots diminished. Microscopically, the chief changes to be observed are a diminution of motor ganglion cells and of axis cylinders. The multipolar cells have disappeared, or are represented here and there by a dwarfed and pigmented cell which has lost its processes. Similarly the motor nerve fibres have disappeared, or only remnants exist in the form of a few shrivelled axis cylinders. All the rest have been destroyed by the pressure effects of increased inflammatory connective tissue.

The palsied muscles are limp and atrophied. Their muscular fibres are also attenuated; they lose their transverse striation and become granular; whilst the connective tissue between the bundles is considerably increased.

Symptoms.—The onset is sudden, and is characterised by access of fever, with, it may be, convulsions; after which there is found paralysis of muscles in the limbs, or often only in one limb, followed by their rapid impairment and wasting. Eventually, arrest of development of bones, and various consequent distortions occur.

A frequent history of a case is as follows. The access, as stated, is sudden. The child may be teething, or it may be found to be 'feverish,' or it may actually be recovering from

some specific fever. It is put to bed without causing any great anxiety, until when taken up in the morning, after its sleep, it is found to be weak in one of its arms or legs.

Or the attack may cause alarm from the first, by the presence of vomiting, convulsions, and perhaps coma. The palsy may be found to have affected the arms or the legs, or a single limb, or to be limited to a group of muscles; but the paralysis is complete from the first and extends no further. The affected extremity is powerless; the muscles involved are limp and flaccid; they waste rapidly and lose their electrical (faradic) sensibility; the extent of this latter, however, varying according to the amount of the damage to the motor cells. The deep reflexes are lost; but there is no loss of control over the sphincters, no pain, no anaesthesia, as a rule, and no tendency to bedsores. The above conditions constitute the first stage. The limb, however, recovers except in the certain involved muscles or groups of muscles.

After about six months the palsied muscles are found almost completely atrophied and shrunken; the superficial temperature over them is markedly diminished, owing to lessened blood supply from narrowed calibre of their blood-vessels; the bones are arrested in their development, and remaining somewhat softer than normal, they become bent and distorted by the unresisted action of the healthy muscles. Consequently we find different varieties of club-hand and club-foot, talipes equinus being the most frequent. As regards the lower limbs, it is remarkable how the muscles below the knee are almost invariably involved in preference to those above the joint.

Occasionally some of the muscles of the neck or trunk are affected, but rarely so as compared with the extremities. Relapses or fresh outbreaks are not unknown.

Diagnosis.—(1) FROM RICKETS.—In this disease, although the child may not be able to stand, owing to weakened muscles, it can still move its limbs. Further examination will reveal deformities of chest and skull.

(2) FROM PERIPHERAL NEURITIS.—This disease is less acute than infantile paralysis. Further, if on carefully noting the distribution of the paralysis it is found that those

muscles are affected which are supplied by one nerve, it is due to a peripheral lesion ; but if muscles are affected which are supplied by different nerves, then the lesion must be in the anterior cornua. Again, if after the paralysis begins to abate, and muscles in different parts begin to waste and lose their faradic irritability, it can only be due to anterior polio-myelitis.

(3) VOMITING in a child should never be overlooked. It may cause prostration, but never total immobility of muscles.

Prognosis.—The disease itself is rarely fatal, and the outlook as regards life is therefore favourable. The prognosis on the question of muscular recovery depends entirely on the response to electrical stimulus. Thus—(i) those muscles which respond readily, tend to recover without aid ; (ii) in those in which the electrical contractility is feeble, the recovery is doubtful but still hopeful ; (iii) those in which no response can be elicited are probably beyond treatment.

Treatment.—During the onset we should direct our attention towards allaying all feverish symptoms and preventing any further extension of the morbid process. Keep the child quiet in bed. Give a purgative (calomel) to expel any irritant in the intestines. Belladonna or hyoscyamus may be given, with a view to arrest extension of the disease. But their influence is probably over-rated. Absolute rest is still essential (even should feverish signs have abated) for one or two weeks, or until the case may be looked upon as quiescent. Then give cod-liver oil, Parrish's food, or steel wine, and such good nourishing food as is calculated to improve the general nutrition of the child, and so help the natural process of repair in the spinal cord. When there is no fear that strychnia will act as an irritant on the cord, but rather as a stimulus, it may be given cautiously and in small doses. It is then of great service, but prior to this stage it may do harm.

But most cases, unfortunately, do not respond favourably to this treatment. Then electricity must be applied ; nor should it be delayed, lest the atrophied muscles reach a condition from which they can never recover. At the same time,

its improper application may do harm. So long as the trophic function of cord and of the nerves to the muscles is disorganised, as shown by fever and irritability, electrical stimulus will increase the damage. If the cornual cells and the motor nerve have both been destroyed, no stimulation can do any good; but if damaged only, an impulse may after a time be conveyed, and, by its influence on the muscular fibre, ultimately lead to the latter's regeneration. If then a case be quiescent, and if after repeated rubbings, shampooings, and other forms of friction, the affected muscles show no sign of recovery, then we must resort to electricity without delay. The constant current is best; one terminal being placed at the site of entrance of the nerve into the muscle, the other gently impressed over the muscle at various points. Weak currents only should be used at first, and afterwards gradually increased. Nor need any current be applied to those muscles which, although enfeebled, still retain their faradic irritability. The use of electricity should be continued for some months or even a year, as even if it fail to restore muscular power, it at least delays the deformities. Additional help may subsequently be afforded by the exhibition of strychnia, iron, and cod-liver oil.

ADULT SPINAL PARALYSIS

Symptoms.—In this disease the symptoms and onset are much the same as in the infantile variety. It commences somewhat abruptly, with febrility, and is followed by complete paralysis of one or more limbs, or of certain muscles in a limb. The muscles then waste, lose their electrical irritability, and then, after a time, tend to recovery or lapse into complete destruction. It should be remembered, however, that the disease is less acute in the adult, and also that arrest of development of bones is obviously not a marked symptom, and that deformities are consequently less pronounced. Usually the lower limbs are affected in preference to, and almost always before, the upper.

Pathology.—As in the acute infantile disease.

Treatment.—The same general principles will also obtain

as in infantile paralysis. Galvanic stimulation can be carried further, and with greater intensity, than in children.

SUB-ACUTE ANTERIOR POLIO-MYELITIS

(CHRONIC GENERAL SPINAL PARALYSIS)

Definition.—A chronic inflammatory disease of the anterior cornua, characterised by weakness of legs and thighs in that order, and subsequently of the forearms and arms.

Pathology.—Practically the same as in infantile paralysis ; but the disease is more chronic, and the destructive changes less marked.

Symptoms.—The disease is an essentially chronic one in its clinical aspect. It begins gradually ; and instead of being arrested as in the acute form, it progresses from bad to worse. The first symptoms are observed in the lower limbs as a rule, although, rarely, it has been known to commence in the upper. Thence it extends to the forearms and arms. The muscles which are affected first become paralysed, they then rapidly waste, and as the lesion is in the anterior cornua of grey matter, the ‘reaction of degeneration’ is soon apparent.

Usually the anterior tibial group is first affected ; then the flexors of the thigh and the extensors of the knee become involved in that order. The paralysis, however, is not supreme at first ; on the other hand, it tends to increase, and not to amelioration.

The superficial and deep reflexes are abolished early ; but there are no rigidities, no tremors, no sensory disturbances, no affection of bladder or rectum, and no fever.

Subsequently a similar condition affects the muscles of the forearm, usually beginning in the hand muscles, afterwards affecting the extensors of the wrist and fingers, and thence extending to the other muscles of the upper limb. Then, as the disease progresses, the muscles of the trunk, or of the neck, or of those which are supplied by the motor cranial nerves, become implicated ; and in them, when once attacked, there is progressive advancement, and no amelioration or even arrest, until eventually the limbs and body are so attenuated

as to resemble a patient in the last stage of chronic phthisis. Then, and then only, may the disease appear to be spent, leaving the patient exhausted, helpless, and bedridden. Partial recovery may take place from this date, unless there are symptoms of failure of the respiratory, or cardiac, or other medullary functions.

Diagnosis.—(1) FROM ACUTE ANTERIOR POLIO-MYELITIS.—In this disease the onset is sudden and febrile, and at once reaches its acme of severity.

(2) FROM PROGRESSIVE MUSCULAR ATROPHY (Chronic Anterior Polio-myelitis).—Here the paralysis usually commences in the hands (thenar eminences) ; it is less acute, and the atrophy precedes, and is responsible for the severity of, the paralysis.

(3) FROM PERIPHERAL NEURITIS, see p. 720.

Treatment.—No medicinal treatment is known to be of any service. We may try the effect of strychnia, or of the many compounds of iron, arsenic, zinc, and other metals which have a reputation in nervous lesions. It is probable, however, that when recovery takes place after a course, say of arsenic, it would have taken place equally soon without the drug.

The best results would seem to follow the regular and persistent use of galvanism, together with massage and passive movements of the affected limbs.

CHRONIC ANTERIOR POLIO-MYELITIS (PROGRESSIVE MUSCULAR ATROPHY)

Definition.—A chronic inflammatory disease of the anterior cornua, characterised by progressive muscular atrophy, which first attacks the thenar group.

Causation.—*Age* has apparently no influence ; it may occur at all periods. In our experience, we have found it most frequent in middle life. *Sex.*—Most cases occur in males. *Heredity.*—A strongly-marked feature. It may occur as a neurosis in the offspring of parents who suffered from some other form of nerve disease ; or it may actually descend from

father to son, or from mother to daughter. *Occupations*.—Those which involve prolonged muscular strain on a given set of muscles. Thus we have seen it in a mechanic whose chief duty was to ‘hold up’ against rivets which were being clinched by repeated hammerings. Again, another patient’s duties consisted of mowing a large lawn with a machine ; hence his hands were subject to constant strain and muscular exertion. *Injury and Exposures*, also *Mental Emotions and Worry*, are predisposing causes.

Pathology.—The lesion is situated in the anterior cornua of grey matter, and in the motor roots issuing therefrom. The change, which is essentially an inflammatory one, commences in the anterior group of multipolar ganglion cells. These lose their processes, shrink, become pigmented, and finally disappear. Sclerotic changes also occur in the tissue of the anterior cornua which surrounds these cells, and ultimately extends to the anterior nerve roots, the tubular fibres of which atrophy, shrink, and lose their insulating fatty sheaths. The muscles which are affected chiefly show fatty degenerative changes in their fasciculi ; they lose their transverse striæ, the sarcolemma sheaths being filled with fat, or remaining as fibrous tissue.

Symptoms.—The chief symptom is chronic and progressive wasting of muscles, remarkable for its symmetry. The onset is gradual ; and although different parts of the body may each be first to show the atrophic change, its most usual site for commencement is in the muscles of the thumb of the right hand. The thenar eminence dwindles, and instead of being plump and rounded, becomes shrunken and excavated. Subsequently the left thenar eminence undergoes a similar change ; and whilst this is in progress, atrophy takes place in the hyperthenar eminence and in the interossei of the right hand, the left hand following suit shortly after. Hence the hands assume a characteristic claw-like expression. From the hands the atrophy extends to the muscles of the forearm, but apparently in no recognised sequence. Then the muscles of the upper arm are involved, especially those attached to the scapula. Subsequently the muscles of the head and neck are affected, and also those of the back, abdomen, and pelvis ; but,

singularly, the urinary and rectal muscles escape. The legs are affected later on. Occasionally the disease commences in the lower extremities ; but this is rare. Should it commence in the lower extremities, the peronei are the first muscles affected, thus producing a talipes varus ; occasionally, however, the calf set of muscles shows the commencement with characteristic deformity (t. calcaneus) ; thence it usually spreads to the trunk and hand muscles. In children the muscles of the face often get involved, producing an idiotic expression.

In addition to the wasting of muscles, there is also loss of power, the weakness following, and being due to the atrophy, and in proportion to it. But there is no complete paralysis until all the muscular fibres are destroyed. Similarly, the faradic contractility of the muscles is not impaired, or at least not absent, until their fibres are completely lost ; nor is there impairment of sensation. In addition, certain fibrillar twitchings may be from time to time observed in the muscles which are undergoing change. This is specially seen in the ball of the thumb, and in other muscles covered by thin skin only. It can be usually elicited by gently striking the muscles with the handle of a pen or some other light object. Similar but more marked muscular convulsions may affect the muscles of the arms and legs, and then they produce the grosser movements of flexion or extension.

The further course of the disease varies in many cases. The progress may be very slow, or arrested for a time, and then advance more rapidly ; or death may occur within a comparative short illness, from involvement of the muscles concerned in deglutition, respiration, or other vital function.

Diagnosis.—(1) FROM LEAD PALSY.—In this disease the paralysis comes on suddenly, and is out of all proportion to the comparatively small amount of atrophy. The patient improves under treatment (iodide of potassium and strychnia). Seek for the blue line on the gums.

(2) FROM ACUTE ANTERIOR POLIO-MYELITIS, see p. 686.

(3) FROM PARALYSIS OF ULNAR NERVE.—Here the disease would not be symmetrical ; and there would be loss of sensation, as well as of muscular power, in the parts supplied by the

nerve. The appearance of the hand, however (*main en griffe*), would be almost identical with that of progressive muscular atrophy.

(4) WRITER'S CRAMP.—The atrophy is limited to the hands, or even to one hand, and proceeds no further. Improvement takes place under rest and galvanism. (See Suckling, *op. cit.*)

Treatment.—Rest generally, and especially to the muscles involved. In electricity appears our chief hope. The muscles affected should be stimulated daily for fifteen minutes. Faradisation is best. The strength of current will depend on the amount of atrophy; the more advanced the wasting, the more intense should be the stimulation. Give iodides freely if a syphilitic origin be suspected. The patient should be warmly clad, and his food should be nutritious.

LOCOMOTOR ATAXIA

Definition.—A progressive disease, due to sclerosis of the posterior columns of the spinal cord, characterised by loss of co-ordination of muscles, without loss of muscular power, commencing usually in the lower extremities, and gradually extending to other parts of the body.

Causation.—(1) *Age.*—From twenty-five to forty-five. (2) *Sex.*—Males, in ratio of about 80 per cent. (3) *Syphilis*, in over 80 per cent. of cases (Erb); this, as a factor, is probably exaggerated. (4) It may be *hereditary*. (5) *Nervous Exhaustion*, fatigue, anxiety, and exposure. (6) Occasionally *secondary* to some acute specific fever. Probably the most potent causal factor is a combination of syphilis with venereal excesses.

Pathology.—Sclerosis of the posterior root-zones (columns of Burdach) is the essential lesion (Charcot); but the change may extend into the columns of Goll, and even into the lateral columns. The lesion is more marked and extensive in the lumbar region; but as it ascends towards the cervical region, it involves the columns of Goll. The membranes of the cord are congested and often adherent to the cord at the posterior aspect.

The change is more of the character of a degeneration than of an inflammation.

The centripetal nerves coming from the muscles are therefore either cut off from, or disorganised in their passage to, the ganglion cells of the anterior cornua, from which the centrifugal nerves issue. Hence there is a break in the circuit, with the result that the muscles lose their co-ordination and their concerted movements, although they do not waste. Pain is, of course, easily accounted for by the mechanical effects of the injury to the posterior nerve roots.

Symptoms.—The presence of three signs alone are diagnostic, viz. : loss of knee-jerks, lightning pains in the limbs, and contracted pupils, which are insensible to light, but act to accommodation.

But there is no regular order of symptoms ; incoordination of muscular movements concerned in locomotion, or girdle pains, or vomiting, may each appear early. Symptoms may therefore be classified or grouped as follows :

(1) PAINS of greater or less severity darting through the body, or down the thighs or arms (lightning pain), occurring at varying intervals. Or they may be of a constrictive character, mostly round the upper abdominal region (girdle pain). Or they may be lancinating or gnawing in the lumbar, calf, or other groups of muscles. The pains are usually accompanied by cramps, showing that the lesion is situated outside the grey matter of the cord, *i.e.* in the posterior root-zone. The muscles do not lose power, nor are they wasted. Occasionally there is loss of sensation in various parts.

(2) DISORDERS OF VISCERAL FUNCTIONS.—*Stomach.*—Violent attacks of indigestion ; pains commencing in the groins and extending to epigastrium, accompanied by severe vomiting, which occasionally relieves the pain (gastric crises).

Rectum.—Tenesmus, associated with a bursting sensation in the rectum, and occasionally involuntary passing of faeces ; but paralysis of sphincters is rare.

Bladder.—Dysuria, with frequent desire to micturate ; vesical catarrh ; urethral pains.

Heart.—Acceleration of pulse, especially during the gastric

crises; or irregularity, or dirotism. Anginoid attacks, not infrequently associated with valvular disease.

(3) AFFECTIONS OF SPECIAL ORGANS.—*Eye*.—Colour-blindness, diplopia, flashes of light, contracted field of vision, contracted pupils, which respond to accommodation but not to light (Argyll-Robertson pupil), various squints, or proptosis, blurred definitions of near and distant objects, with, finally, amaurosis (blindness) due to white atrophy of the optic discs. Occasionally this eye symptom precedes all others. *Ear*.—Deafness is frequent, and often attended by ‘buzzings,’ ‘drummings,’ and other distressing phenomena. *Sexual Organs*.—Virility, increased in early stages; totally lost, with spermatorrhœa and incontinence of urine later on. *Larynx*.—Spasm of muscles, causing dyspnoea or cough. Occasionally paralysis of phonatory muscles.

(4) TROPHIC DERANGEMENTS.—*Perforating ulcer* of foot, which may be one of the earliest symptoms. Various disorders of *skin*, such as cutis anserina, eczema, herpes, shedding of nails. Atrophy of *muscles*, which follows no regular or progressive order, and which occurs only in advanced stages. *Joint* troubles; arthritis (Charcot’s disease) commencing with synovial effusion, which is painless and without rise of temperature, and is followed by erosion of the heads of bones, and finally by dislocations. The knee joint is most frequently and earliest affected; the joints of the upper limbs are not usually involved until very late (see Rheumatoid Arthritis). Fractures may occur in the prodromal stage, and always in the vicinity of joints, and are due to atrophy of bones (Rivington).

(5) MUSCULAR INCOORDINATION.—The gait is the most characteristic symptom. When starting to walk, the legs are thrown forwards and outwards with sudden jerks, the heels are banged down on the floor with unnecessary violence. The patient cannot walk without assistance; he directs his movements of legs by fixing his eyes on the ground. In other words, his loss of coordination produces the puppet-like gait of Trousseau. His walk has, again, by others, been likened to the ‘goose-step’ and to the piper’s strut. When started, he keeps going and cannot turn without stopping. He uses his

arms as a balancing pole. When standing, he totters and cannot maintain his equilibrium if he places his feet together, especially if he shuts his eyes; the body is bent forward, in order to counteract strong contraction of the extensor muscles, which by themselves would cause the patient to fall backwards. He has a sensation as though walking on cork socks or wool. When the disease has affected the higher parts of the cord, similar defects of coordination in the hands and arms are observed, but less intense. He has difficulty in mounting stairs, hanging his hat on a peg, or even feeding himself. In later stages there occur spasmodic contractions of muscles, excited by will, but quite without control when started; therefore his movements are hurried and precipitate. These signs are best marked in the lower limbs, and may extend no further.

(6) NERVOUS DISORDERS.—Anæsthesiæ of different areas are common. In the later conditions, paralysis of third, fourth, sixth, seventh, and other cranial nerves may supervene. The mental condition is usually unimpaired to the end; but occasionally insanity, in one or other form, closes the chapter.

Rarely, the disease commences in the upper extremities, with ataxy (see diag. 29) and loss of muscular sense, as in the lower limbs.

Diagnosis.—(1) FROM CEREBELLAR DISEASE.—Here there is staggering, but no jerky gait. There are also frequent vomitings and convulsions, with double optic neuritis.

(2) IN HYSTERICAL ATAXY, the incoordination only occurs when the patient is standing, and does not happen when she is lying down. Further, she has no pains, and the knee-jerks are exaggerated.

(3) IN SYPHILITIC DISEASE, the pains and other symptoms are cured or relieved by iodide of potassium.

(4) FROM MULTIPLE NEURITIS, see p. 720.

The absence of knee-jerks, associated with lightning and girdle pains, and the Argyll-Robertson pupil, are sufficient data on which to found a diagnosis. (See Suckling, 'Diagnosis of Diseases of the Nervous System.')

Prognosis.—Unfavourable. Death occurs in four or five years, generally from exhaustion. Occasionally, however, life may be prolonged for ten years or longer.

Treatment.—Mainly palliative. Cases of recovery have been recorded. Give iodides if a syphilitic origin be suspected. Nitrate of silver (gr. $\frac{1}{3}$), phosphorus (gr. $\frac{1}{20}$), and pilocarpin (gr. $\frac{1}{10}$) at are times beneficial. Symptoms are often relieved by cold baths, rubbings, and massage; also by various forms of electricity. Give opium, hyoscyamus, or Indian hemp, to assuage the pains. Nerve-stretching (by operation), especially of the great sciatic, has recently been advocated, in order to relieve the special lightning pains; but the results appear to be temporary only.

Suspension by the neck may be practised, with a view to relieve the pains, gastric crises, and other distressing symptoms. The results appear to have been variable, success attending some cases, failure occurring in others. In our own experience of three or four cases, relief certainly was obtained, but it was fugitive only. On similar grounds, forced flexion of the thigh on the abdomen, to stretch the great sciatic nerve, has been tried.

Stomach, rectal, and bladder crises are best treated locally. Strychnia should be avoided (Ross). Gowers recommends chloride of aluminium (grs. 1 to 4).

Beyond this, we should order the patient to wear warm clothing, to have plain, simple, and easily-digested food. He should also guard against constipation.

HEREDITARY ATAXY (FRIEDREICH'S DISEASE)

Definition.—A congenital disease of the posterior and lateral columns of the cord, characterised by muscular incoordination, tremors, and a hesitating character of speech.

Causation.—Nothing is known of any predisposing causes of the disease, except that, a case appearing in one child, others of the same family may sooner or later show signs of the disease. The disease thus appears to be transmitted from parent to offspring, but without the parents being affected. Either boys.

or girls may be attacked ; but it apparently, in a given family, tends to affect the boys only, or the girls only.

The first symptoms appear between the commencement of permanent dentition and puberty.

Pathology.—A sclerotic lesion is found invading the whole of the posterior, and extending into the lateral columns. In the lateral columns the disease involves not only the crossed pyramidal, but also the cerebellar tracts. The posterior nerve roots are also usually affected.

Symptoms.—The first symptoms observed are those of ataxy, and nothing else. There is a marked and jerky inco-ordination of movements in the legs. This then extends somewhat rapidly to the trunk, arms, and head and neck. Subsequently the symptoms resemble somewhat a combination of chorea, disseminated and lateral sclerosis. That is to say, the muscles become weakened ; the head is tremulous and unsteady ; the speech is hesitating, stammering, and thick, apparently from muscular incoordination ; there is nystagmus, especially in the late stages ; and the knee-jerks are increased, although the superficial reflexes are diminished. On this latter point, however, there are differences of opinion, many authors insisting that the knee-jerk is absent. This symptom will probably vary according to the extent of the disease in the lateral columns, as compared with the posterior root-zones.

With all these symptoms, the pupils are equal, and respond to light and accommodation ; the discs are normal ; and there are no visceral crises, and no joint or bone lesions. Nor does the patient complain of girdle or lightning pains ; but cutaneous sensibility is duller than usual.

The disease is a progressive one, and eventually the muscles of the limbs become much enfeebled, and also those of the back, so that a characteristic curvature of the spinal column is developed.

Prognosis.—Unfavourable. Yet the patient may survive for a number of years, the disease often remaining stationary. Death is usually due to some accidental disease.

Diagnosis.—FROM LOCOMOTOR ATAXY.—In Friedreich's

disease there is an absence of the characteristic lightning pains and eye symptoms.

FROM DISSEMINATED SCLEROSIS.—In this disease the movements are more rhythmical and not so jerky. The speech is not so thick, but is more syllabic. Further, the tremors begin in one limb and then extend to the opposite one.

Treatment.—No medicines have any beneficial result. Those palliative remedies may be tried which are mentioned under Locomotor Ataxy.

LATERAL SCLEROSIS

Definition.—A term which includes at least three different conditions or diseases, which are characterised by sclerotic changes occurring in the lateral columns of the cord (crossed pyramidal tracts), by rigidity of limbs, and by increase of superficial and deep reflexes.

(1) Primary Lateral Sclerosis

Causation.—*Age.*—Most frequent in early adult life. *Sex.*—Men mostly ; it is comparatively rare in women. Repeated exposure to *cold, wet*, or to *blows and injuries* to the spinal column and cord. *Heredity* exerts no direct influence, except that in some cases there is an inherited neuropathic tendency (insanity, epilepsy, &c.). *Arthritis.*—It has been known to supervene on traumatism to joints, and also after gouty arthritis.

Pathology.—The sclerotic change is found in the lateral tracts. Both sides are affected symmetrically and simultaneously. The lesion may extend to the lateral surfaces of the cord, and even back to the posterior horns of grey matter.

Symptoms begin gradually. A sense of weakness commences in the legs : they feel tired and heavy ; then they become rigid, or the rigidity and loss of power are contemporary, and advance together.

As a result of these conditions, the patient's gait is characteristic. He has difficulty in walking ; he requires the help of a stick ; he shuffles along, his toes scraping the ground. His

deep reflexes are exaggerated, ankle clonus and patellar clonus being well marked. Similarly, the slightest stimulus is enough to elicit any of his superficial reflexes. But with all these there is no muscular wasting and no pain; nor has he any loss of tactile sensation, nor of control over bladder and rectum.

As the disease becomes more chronic, the rigidities increase. The heels may be violently drawn up, so that the toes point to the ground (*talipes equinus*); or the powerful adductor muscles may draw the whole limb across the middle line. On forcibly raising one limb from the couch, the spasm may be so marked as to extend to the opposite leg, and hence both limbs are raised at once. On attempting to walk, the patient appears to have no joints; the legs move as a whole. On setting the foot down, the stimulus is enough to excite violent twitchings (*clonus*) in the calf and other muscular groups. Occasionally this spasmodic condition may affect the flexor muscles instead of the extensors.

When the paralysis is complete, the rigidity is also complete and permanent; so that a joint (say the knee) cannot be flexed or extended without considerable force and difficulty. Further, the muscles, after the slightest irritation or stimulus, are liable to prolonged paroxysms of convulsive tremors.

The symptoms are not necessarily limited to the lower limbs. The arms may be affected secondarily; or the disease may, at times, first show itself in the upper extremities. Occasionally, indeed, the muscles of mastication and deglutition are involved.

Diagnosis.—From CHRONIC MYELITIS due to caries of bone; or to the presence of spinal tumour.

In this lesion the weakness precedes rigidity, and there are usually sensory disturbances, together with bladder and rectal troubles.

Prognosis.—The disease is essentially chronic and progressive; it may be arrested; and cases of recovery have been recorded. Of itself, it has little tendency to shorten life. Death usually occurs from exhaustion, or from medullary complications. Bladder and secondary kidney affections are

rare, probably owing to the excessive reflex action preventing retention and cystitis.

Treatment.—Unsatisfactory. Prolonged rest in bed gives the best results. We may also resort to gentle friction and shampooings. These even should be discontinued if they excite muscular spasm. As regards medicine, a long course of arsenic appears to give the best results. Strychnia should be avoided, as it increases muscular contractions.

(2) Amyotrophic Lateral Sclerosis

Causation.—*Age.*—It occurs most frequently in adult life.

Sex.—Women, in the proportion of quite three to one.

Occupation.—It would appear, from notes of a series of four cases which we have observed, that the disease is associated with occupations entailing muscular strain and exertion of a severe character. For example, we have seen it in female acrobats and trapeze performers (two cases); another case occurred in a horse-breaker; whilst the fourth patient was a woman who had been engaged in the arduous work of chain-making. The other predisposing causes would be the same as in primary lateral sclerosis.

Pathology.—The sclerotic change here is also found in the crossed pyramidal tracts; but it is not limited to them: it extends to the anterior horns of grey matter. It is a combination of disease of the lateral tracts and anterior cornua. It usually commences in the upper part of the cord, and proceeds downwards. It also frequently extends to the medulla, producing fatal bulbar symptoms.

Symptoms.—The disease also begins insidiously, as in primary lateral sclerosis. Indeed, the symptoms resemble that form in most of their characters, except that the upper limbs are the first to become involved. Usually there is a precedent numbness or tingling in the arms or hands. Then the muscles become enfeebled and rigid, and eventually they emaciate. The emaciation, however, is subsequent on the paresis.

It does not affect muscles in groups; nor does it 'creep' from one to another, but attacks all the muscles of a limb at

the same time. As a result of the rigidity, the arms are fixed to the side, the forearms flexed and pronated, and the fist clenched.

Subsequently weakness and rigidity of the muscles of the lower extremities supervene, and possibly emaciation also. The emaciation, however, is not so marked a feature as in the arms.

As in primary lateral sclerosis, the reflexes are exaggerated in the early stages; the muscles are subject to violent clonic spasms on the slightest irritation; but there is also no affection of bladder or rectum.

The subsequent progress of the case is marked by extension to the medulla, causing paralysis of tongue, lips, and face, together with symptoms peculiar to bulbar disease. The phrenic nerves may also be involved in the upward course of the disease, with consequent affection of the diaphragm.

Prognosis.—The disease is invariably fatal, death occurring usually within three years from the onset. The immediate cause of death is due to implication of the motor nuclei of the medulla.

Diagnosis.—It may be confounded with (a) PROGRESSIVE MUSCULAR ATROPHY. In this disease, however, the muscular atrophy is confined to groups, or even to a single muscle; there is an absence of rigidities and increased reflexes, and consequently of spastic paraplegic gait.

(b) From PRIMARY LATERAL SCLEROSIS it is distinguished by the general wasting of muscles due to changes in the anterior cornua, and by the medullary symptoms, all of which are absent in the former disease.

(c) In DISSEMINATED SCLEROSIS the early signs may resemble those of amyotrophic lateral sclerosis if the cord lesions extend to the anterior cornua and lateral tracts; but eventually the diagnostic scanning speech and nystagmus would supervene.

Treatment.—Attend to the general health. Let the patient live in the open air as much as possible. Faradisation to the affected limbs may be of some avail. Iodides, with or without mercurials, may be given if any syphilitic taint be

suspected ; indeed, these remedies are often prescribed when there is no evidence whatever of syphilis. Arsenic, in increasing doses, appears to afford the best results. But, at most, medicinal treatment is of little avail.

(3) Secondary Lateral Sclerosis

Causation.—(1) Some *injury to the motor centres of the brain*, or to the fibres below converging therefrom, such as hæmorrhage (apoplexy), embolism, tumours, or a patch of softening.

(2) *In infants*, either from an arrest of development of the motor area, or from direct injury caused by forceps delivery, or by a fall or blow.

(3) *Paraplegia*, owing to degenerative changes occurring in the cord subsequent to the primary lesion.

Pathology.—Sclerotic changes are found in the crossed pyramidal tract, usually one-sided, unless there are multiple causal lesions in the higher centres. As the lesion is traced down towards the lower extremity of the cord, it occupies a smaller area in the lateral column, and is situated nearer the posterior horn. Occasionally, but rarely, the sclerotic changes extend to the anterior horn, and to the direct pyramidal tract (fasciculus of Türck).

Symptoms.—They resemble in a marked degree all those symptoms described in the primary form, except that the disease is not a primary affection of the cord ; and therefore, although the onset of the symptoms is gradual and insidious, it is anticipated by one or other of the lesions enumerated above ; and it is consequently usually unilateral.

Rigidity of limbs is an early sign. The arm and leg are stiffened and adducted ; the thigh is frequently drawn across the middle line ; the foot is distorted usually in the form of talipes equino-varus ; the fingers and wrist are strongly flexed, and the forearm bent and pronated. Tremors of muscles are well marked, especially after forcible movements of the limb ; the deep reflexes are increased.

Further, if the pathological changes extend to the anterior horn of grey matter, wasting and degeneration ensue in those

muscles receiving their nutrition from the area involved. In that variety which ensues on hemiplegia, various choreic movements are apt to take place in the arm ; or the convulsions may be so marked as to cause the wildest excursions of the upper limb, entailing the necessity of strapping or otherwise fixing it to the side of the body (mad arm). Or, again, the movements may be limited to irregular twitchings of the fingers (athetosis).

Treatment.—No special rules can be given, beyond those which are described under the other forms of lateral sclerosis. As the condition is entirely a secondary one, and usually dependent on some cerebral lesion, the question of surgical procedures may have to be discussed.

SPINAL TUMOURS

Definition.—Morbid growths originating in the cord itself, its membranes, or its bony canal. Under this heading would also be included parasitic invasion, aneurysm, &c.

Pathology.—Different forms of new growth may involve the neural canal and its contents. The most frequent, however, are syphilis, tubercle, malignant new growths (sarcoma, carcinoma), fatty tumours. Hydatid cysts, though not unknown, are comparatively rare. Further, injuries (such as caries, dislocations) may, by their results, produce symptoms identical with those of tumour. A similar remark applies to hæmorrhage. It must also be borne in mind that a tumour may exist in the canal without causing symptoms until it is suddenly roused to activity by some blow or injury.

Tubercular growth occurs most frequently in early life, syphilis in middle life, whilst malignant new growths are most frequent in the aged.

Growths in the cord itself are mostly syphilitic, or gliomatous, and are frequently single. Those springing from the dura mater are generally syphilitic, tubercular, or sarcomatous, and are more often multiple. Those originating in the bony canal itself are usually, but not invariably, single.

Symptoms.—In all tumours affecting the cord and its

membranes, there are many signs and symptoms in common. Thus there is more or less complete motor paralysis of parts below the lesion ; and symptoms, such as hyperæsthesia or anæsthesia, pointing to compression of the sensory nerve roots. The more exact details, however, vary, not only according to the level and situation of the lesions, but also according to the transverse extent of the affection of the cord. Further, the acuteness of the symptoms, and the order or date of their onset, often vary in the different diseases which cause them.

Thus paraplegia may involve the legs only, in lumbar lesions ; or it may affect the arms and legs if the tumour be situated in the cervical region. Its onset may be gradual in its development in slowly growing tumours ; or it may be sudden, with remissions and exacerbations, when it is due to inflammatory extensions from the primary growth to the cord. Again, the pressure on the cord may be confined to certain columns and tracts, and give rise to symptoms resembling locomotor ataxia, lateral sclerosis, or ascending sclerosis, and the like.

Pain is not necessarily a marked symptom ; indeed, when the new growth develops in the grey matter of the cord, it may be absent until it has extended to the surface. On the other hand, pain may be intense and agonising in those cases where the tumour is developed on the surface of the cord, and especially in those cases where the cord is compressed antero-posteriorly ; or when a new growth or aneurysm encroaches on the subdural space or vertebræ. The most agonising pain we have witnessed occurred in a case in which sarcomatous growths were found existing between the dura mater and vertebral laminæ. But pain, even when present, does not always persist. Sensation may soon be entirely lost when the posterior nerve roots become destroyed. The advent of pain is frequently marked by premonitory numbness and tinglings ; or by herpetic or bullous eruptions in the course of irritated nerves.

The reflexes, as a rule, are exaggerated.

It must be remembered, however, that there are no definite and constant signs of spinal tumour. A growth may exist for

months before it affords signs of pressure ; but afterwards, the course of events is more rapid. If, however, according to Sharkey (Galstonian Lectures, R.C.P., 1886), a tumour arise *within* the cord, it produces disturbance of function of cord from the first ; but it may not produce striking pathological phenomena until the cord is grossly compressed against its bony canal, or the elasticity of its membranes is exhausted.

Diagnosis.—The diagnosis of spinal tumour must depend on the earlier symptoms of its onset, if they can be obtained. These would be, in the main, the signs of a localised pressure, producing gradual paralysis of the nerves involved ; and eventually extending to paraplegia, accompanied or preceded by severe pains. The temperature is not necessarily increased. The diagnosis of the character of the tumour would be assisted if there be any tubercular history or physical signs of tubercle elsewhere ; or any history of syphilis ; or if there be any signs of cancer or malignant neoplasm, in any of the organs of the body.

Prognosis.—Generally unfavourable, except in syphilitic cases, or in those in which the tumour is local and discrete, and thus comes within the reach of surgery. Otherwise, death occurs within a time varying from a few months to a year or two ; and may be due to exhaustion, consequent on the effects of cord compression, or to phthisis in tubercular cases, or to amyloid degeneration in syphilitic disease.

Treatment entirely depends on the diagnosis. We may hope for amelioration in syphilitic tumours, by the prolonged administration of iodide of potassium with mercury.

There is one point on which stress must be laid. When a diagnosis of syphilis shall have been arrived at, antisyphilitic measures should be given without an hour's delay, and with such vigour as to produce speedy constitutional effects. The cord is quickly damaged, and with difficulty restored.

Cod-liver oil and other remedies suitable for tubercular disease are indicated in a tubercular diagnosis.

Many tumours are amenable to surgical operation (laminectomy). And two essentials must be urged here—viz. the operation should never be delayed and it must be performed

under rigid antisepsis. Cases occur which could have been, in all probability, completely restored to health, had surgery been given a free hand at a date prior to the tumour causing destructive injury of the spinal cord.

SPINAL HÆMORRHAGE

Definition.—Hæmorrhage into the substance of the cord, or within its membranes, or between the membranes and the vertebral canal.

Causation.—Hæmorrhage may be a direct result of *injury* to the vertebral column ; it may be due to the rupture of an *aneurysm* (thoracic aorta, vertebral artery) ; or rupture of blood-vessels, consequent on *muscular spasm* (tetanus), or prolonged muscular exertion. It may also be secondary to certain specific fevers in which hæmorrhages are common (*typhus, small-pox, yellow fever*) ; or to purpura. It may also occur in people who are prone to hæmorrhages (*hæmophilia*) ; and, lastly, it may result from acute *inflammation* of the membranes.

Symptoms.—The symptoms are usually such as are due to spinal irritation, and therefore in their main features resemble meningitis ; but their onset is more abrupt, and their violence more severe. There is a sudden onset of paralysis, both motor and sensory. It is also attended by pain in the back, chiefly referred to the site of hæmorrhage, and extending to the parts supplied by the nerves which are irritated at their origins. Thus the patient may complain, in cervical hæmorrhage, of pain extending from the neck down the arms ; or he may have pain running round the chest or abdomen, as a girdle, in dorsal hæmorrhage ; or shooting down the thighs and legs in lumbar hæmorrhage. This is succeeded by irritative spasm of the muscles of the same regions, and also by tonic spasm of the muscles of the back. Afterwards paralysis speedily follows in all parts below the lesion. There is, however, no loss of consciousness, and the mind remains clear to the end. Cystitis, bedsores, and other evidences of trophic changes are frequent. The symptoms, therefore, represent a combination of those found in acute myelitis and in degenerative affections of the cord.

The symptoms are much the same, whether the hæmorrhage be within the spinal cord itself, or on its surface amongst its membranes. There is, however, one point of difference. If the hæmorrhage be intraspinal, paralysis precedes pain ; if it be on the surface, then pain precedes paralysis.

On the other hand, cases may occur in which spinal hæmorrhage is found post-mortem, and no signs were presented during life. Probably, in such, the hæmorrhage occurs during the last few minutes of life, death being too speedy for symptoms to be complained of.

Prognosis.—Extremely bad, especially in cervical hæmorrhage, when, besides the large extent of cord which is cut off, the phrenic nerves are paralysed. The prognosis, however, improves the longer the patient lives, as then there is a possibility of absorption of the effused blood and restoration of the functions of the cord.

Death usually occurs within a few days, or it may be as many hours ; and is often due to arrest of the respiratory function. It may be deferred to a somewhat later period, when it is brought about by secondary inflammation of the cord and its meninges.

Treatment.—Absolute rest is essential. The patient should lie prone on his face, so as to dispose the blood to drain away from the neural canal. Cushions and pillows should be so arranged as to allow of this position with the least distress and fatigue.

Further treatment should be directed to lowering the blood-pressure, either by leeches to the spine or around the anus, or by cuppings, or by free incisions through the skin over the spinous processes, followed by hot fomentations to encourage bleeding. We may also assist this object by strong purgation (calomel, sulphate of magnesia). Ergotin (gr. ij.), subcutaneously injected, may also be tried.

If the hæmorrhage occur in the subdural space, it should be treated as a foreign body, and the spinal canal freely opened antiseptically.

The palsy of muscles, should recovery from the immediate effects of the hæmorrhage promise itself, must be met by electricity, as in meningitis and other similar diseases.

PARAPLEGIA

Definition.—Paralysis of the lower portions of the body. The term is usually applied to paralysis of the lower limbs ; but the arms and trunk may equally well be involved, according to the site of the lesion. It is a symptom rather than a disease.

Causation.—(1) Some lesion of the spinal cord. Paraplegia may originate in disease of the cord itself, or in its membranes, or in the vertebral column. So far as the cord is concerned, there are numerous conditions which will give rise to paraplegia, such as the different varieties of myelitis, simple softening, intramedullary hæmorrhage, and intramedullary tumours. Some of these will have been considered in the various diseases of the spinal cord. A similar remark applies to the different forms of inflammatory affections of the membranes, meningeal hæmorrhages, injuries, and the like, all of which may occasion paraplegia. Lastly, paraplegia may be occasioned by pressure on the cord, as a result of caries of vertebræ, tumours of bone, or of neural canal, and of the spinal nerves which issue from the cord.

(2) Paraplegia may also be a functional disorder, in which no structural disease can be detected. It is met with in hysteria. It may be a reflex condition, subsequent on injury to peripheral nerves ; it occurs secondarily to malaria and anæmia ; and there is also a somewhat rare form of paraplegia dependent upon idea.

The following table, modified after Bramwell, will show the various principal causes at a glance :

1. Organic disease	{	Inflammation of cord	}	Medullary
		Softening " "		
		Hæmorrhage " "		
		Tumours " "		
	{	Meningitis	}	Meningeal
		Meningeal hæmorrhage		
		Injuries		
		Tumours		
	{	Caries of bone	}	Osseous
		Tumours " "		

- | | | |
|---------------|---|--|
| 2. Functional | { | Hysterical
Reflex
Malarial and anæmic
Dependent on idea |
|---------------|---|--|

Symptoms.—The onset may be sudden, or it may be gradual, according to the nature of the causal condition. In an ordinary typical case, the symptoms come on insidiously, with tingling and numbness of the legs and feet. Then follow loss of power, more or less complete; anæsthesia in those parts which are below the lesion; together with constipation, and retention of urine owing to loss of expulsive power in the rectum and bladder.

After a time the sphincters give way, so that the fæces cannot be retained; the urine dribbles away and becomes decomposed, alkaline, and offensive.

At the upper limit of the paralysis there is frequently a band of hyperæsthesia extending round the body, giving rise to a constricting sensation or girdle pain. The paralysed muscles may not lose their electrical irritability; but after a time, if the paraplegia continues, they waste, and exhibit characteristic ‘reaction of degeneration.’ Bedsores, often extensive and deep, are apt to form on the buttocks, hips or heels, and other parts exposed to pressure. The deep reflexes whose centres in the cord are below the lesion are all exaggerated; the skin of the legs becomes dry, harsh, and atrophied, or it may be œdematous.

The above description refers to those cases in which paraplegia is dependent on obvious disease of the cord; and as only those muscles are affected which are supplied from the cord below the lesion, the extent of the paralysis will vary according to the upper level of the disease. Hence in some cases the arms, diaphragm, and trunk muscles are palsied, as well as those of the legs; or again, the disease may involve only one half of the cord (hemi-paraplegia), or it may select certain tracts only, and thereby cause a great variety of paralytic symptoms, according to the columns involved.

As a rule, anæsthesia is more pronounced in paraplegia than in hemiplegia.

Diagnosis.—The diagnosis of the causes of paraplegia due to organic disease of the cord will be found in the various descriptions of myelitis, spinal meningitis, spinal tumour, &c.

Hysterical Paraplegia, the commonest form of hysterical paralysis, would be diagnosed by the suddenness of the paraplegic onset, which, were it due to lesion of the cord itself, could only be caused by violent injury or by hæmorrhage. Such severe lesions as these would be followed by paralysis of sphincters, by the rapid formation of bedsores, and by cystitis and other trophic disorders. The amount of paralysis and of anæsthesia seen in hysteria is quite inconsistent with an absence of trophic changes. We should also be guided by the patient's general symptoms being over-exaggerated, and by her general neurotic condition.

Reflex Paraplegia.—This condition is generally secondary to disease of the genito-urinary organs. The paralysis is not usually complete, some muscles being more affected than others; it also varies from time to time, in accordance with improvement or relapse in the genito-urinary tract. Further, the muscles retain their bulk and healthy reactions; there is not marked paralysis of the sphincters; and recovery takes place when the exciting disease is cured.

In some cases, however, structural changes may extend along the nerves of the bladder or uterus, and so involve the cord; but these can scarcely be regarded as instances of reflex paraplegia.

Malarial Paraplegia is intermittent and periodic. It occasionally represents a true aguish attack, and is cured by quinine or arsenic.

Anæmic Paraplegia.—This may occur after parturition or other severe hæmorrhage. We have seen it supervene in the last days of life, in two cases of slowly rupturing abdominal aneurysm. Moxon attributed ordinary anæmic paraplegia to the precarious blood supply of the lower end of the spinal cord. Hence the paralysis would be more marked in the feet than in the upper parts of the lower limbs.

Paraplegia dependent on idea.—This may occur in males as well as in females, and is not hysterical. From some injury

which is often trivial, a form of paraplegia may ensue. The paralysis, however, is not necessarily paraplegic ; it may be limited to one arm or leg, or to one set of muscles.

The limbs do not waste, except from the effects of prolonged disuse ; there are no bedsores, nor other trophic lesions ; in other words, there is no disease of the spinal cord ; but there is a mental disorder.

Treatment.—Much depends on the cause of the paraplegia. In cases of organic disease, rest is essential ; and it may be supplemented by general sedatives, such as opium and belladonna.

Attend to the patient's general health ; the buttocks and other points liable to pressure should be inspected daily, with a view to the avoidance of bedsores. The urine should be regularly drawn off with a catheter ; and it may be necessary to wash out the bladder with weak solutions of nitrate of silver (gr. $\frac{1}{8}$ ad $\frac{3}{4}$), or of boracic or mineral acids.

Strychnia, iron, and other tonics are required when all signs of irritation have subsided. The attenuated muscles may be restored by galvanism. In paraplegia due to caries of vertebræ, there is good hope of cure by prolonged rest in bed for some months, or, it may be, a year or two.

Other special lines of treatment are described under the various diseases which may cause paraplegia.

PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS

Definition.—A disease characterised by increase of bulk of certain muscles, attended by loss of power.

This disease is considered here only for convenience. No lesion of the spinal cord has been discovered.

Causation.—*Age.*—A disease essentially of late childhood, during the period of rapid development. *Sex.*—Males only ; or at least in the proportion of quite five to one. *Heredity* is often a strongly-marked clinical feature, several boys in the same family being in turn affected. *General Diseases.*—Hereditary syphilis has been thought by some to be a predisposing factor, but without at present sufficient clinical evidence.

Pathology.—No constant pathological changes have been found in the cord, or in the motor centres or nerves. But the muscles have presented remarkable alterations. They are yellow in colour, greasy, and resemble, on section, slices of fat. Microscopically the muscular fibres show not only fatty infiltration, but fatty degeneration of their fibres. Between the component fasciculi, fat-cells are found in excessive abundance, separating the fibres and probably lessening their arterial supply. The fibres themselves are wasted and irregular, being attenuated to a mere thread in some places, and by comparison enlarged in others. Occasionally a muscular fibril is represented only by its sarcolemma sheath ; or again, by fibrous tissue with a few remnants of striped muscular tissue. Even in those parts where muscular tissue is easily recognised, the typical transverse striation is lost or blurred. Whether the disease is a developmental one or merely a degenerative change, is not yet apparent.

Symptoms.—Advice is usually sought for the child, not on account of pain or distress, but because it is ‘weak in its legs.’ It has difficulty in climbing upstairs, and has to raise itself to the upright position by the aid of a chair or other assistance. Then it is noticed that certain muscles of the child’s legs or trunk are abnormally bulky ; at the same time, that its gait is clumsy and waddling ; and that he has apparently spinal curvature (lordosis). Subsequently a similar weakness and enlargement occurs in muscles of the scapulae and upper extremities. Eventually loss of power over the limbs becomes more marked, and is accompanied by distortions and partial dislocations of joints. Then the child becomes bedridden, until, after a miserable existence of three or more years from the initial symptoms, he sinks from prostration, or from intercurrent or accidental disease.

As regards the weakness, it may have been present for some time before it is noticed by either mother or nurse. The child has no method of measuring, and no idea of expressing, its own debility ; but it is present all the same, and is probably overlooked by the mother, who regards her Herculean bantling with pride, and thinks that the lassitude

of weakness is natural to one with such a splendid muscular development.

Certain muscles are almost invariably enlarged, especially those of the calf, the buttocks, and the quadriceps extensors; occasionally the anterior tibial group are increased in bulk. In the upper limb the *infraspinatus*, deltoid, and muscles of the forearm are most frequently enlarged. In another case, it may be the muscular group of the *erector spinæ* which is exaggerated. But there is no necessary order of sequence in the involvement of the muscles; and whilst one set may be increased in bulk in one case, in another sufferer the same group may be wasted. But whether emaciated or enlarged, the muscles are always enfeebled. Certain ones, however, always appear to escape, such as those of the eyeball and face and hands. The pectorals are never enlarged, but often wasted. Those muscles, however, which are small and wasted are feebler than those which are enlarged.

Enfeeblement is always pronounced in the ilio-psoas and in the quadriceps extensor; and this will account for the difficulty in climbing stairs, and also to some extent in rising from the prone to the upright position. There is, however, no loss of control over the emunctories. The affected muscles do not fail to respond to galvanic and faradic currents until they are extremely feeble. Nor is there any 'reaction of degeneration.'

Lordosis is so marked a symptom that the plumb line from the *vertebra prominens* would fall from two to three inches behind the sacrum. It is produced, according to Gowers, by the pelvis and lower lumbar vertebrae being inclined forwards, owing to the weakness of the extensors of the hip, since it disappears when the pelvis is supported on the tuberosities of the ischium, as in sitting.

The gait is a waddling one; the feet are lifted up to unnecessary height, the insteps greatly arched, and then as one foot is planted somewhat helplessly on the ground, considerably wide of the middle line, the weight of the body is lunched on to it, so liberating the opposite foot, which succeeds in a similar manœuvre.

The child has difficulty in rising from the floor. Suppose he is asked to get up when he is on the ground, he raises his trunk by means of an outstretched arm and extended fingers ; the disengaged hand then grasps the corresponding ankle ; having this purchase, the opposite hand grasps its corresponding ankle or leg ; and with alternate shifting of the hands to his knees and thighs he ultimately regains the upright position, by having climbed up himself, as it were.

The later stages are marked by distortion of joints which are usually flexed by the action of the less feeble muscles, the rule obtaining that where muscular enfeeblement is equally distributed, the flexors and pronators overcome the extensors and supinators (see pp. 484, 666).

At this period also, it will be found that the knee and other deep reflexes are diminished or lost, and that the general intelligence is impaired. But sensation is not affected, and it is only in the last stages that the bladder is involved.

Prognosis.—Death usually supervenes in three or four years ; those cases in which the symptoms ensue gradually, usually live longest. Prior to the fatal event, which is generally due to exhaustion or to some inflammatory or specific febrile disease, the patient has been confined to his bed for some months.

Treatment.—No drug is known to exert the least effect in controlling the course of the disease. Nor has electricity in any form had any beneficial effect. The best results have been obtained by carefully regulated gymnastic exercises. The exercises should be so arranged as not to be uselessly expended on muscles which are not affected ; nor should they be too severe or too prolonged. The evidence of powerful men tends to show that muscular strength is cultivated rather by light and easy athletic exercises than by those requiring great efforts. Gymnastics can also be supplemented by shampoos and passive movements of the limbs. Even if no cure be effected by these means, there is considerable arrest in the progress of the disease

LESIONS OF SPINAL NERVES

The spinal nerves, especially those which are superficially placed, or which run in juxtaposition to bone, are liable to various injuries which produce neuritis, and possibly permanent loss of function.

The following examples may be considered as fairly typical of such cases.

The Ulnar Nerve.—This nerve may be influenced by exposure to cold, which produces a neuritis ; or it may be the subject of injury by which the nerve is divided or paralysed. The injury may be situated at the shoulder joint, after a dislocation ; or in the upper arm, from fracture ; or at the elbow, after dislocation ; or near the wrist, usually the result of severe cuts or lacerations.

The nerve supplies the elbow and wrist joints, the flexor carpi ulnaris and half the flexor profundus digitorum, the little and the inner surface of the ring fingers, back and front ; also the muscles of the hypothenar eminence, all the interossei, the adductor pollicis, and the inner head of the flexor brevis pollicis. In other words, it supplies, in the hand, all those deep muscles which are internal to the flexor longus pollicis tendon.

Therefore, when impulses along the nerve are completely cut off, certain characteristic changes occur. Cutaneous sensibility is impaired in the little and the ring fingers, back and front, and perhaps extending to half the middle finger on the back.

Then, in advanced cases, the hand becomes emaciated ; the hypothenar eminence dwindles ; the metacarpal bones are prominent, owing to wasting of the interossei muscles ; the fingers cannot be displayed in abduction ; and the first rows of phalanges are hyperextended by the unopposed extensors ; whilst the second and third rows are semiflexed by the flexors, so that a characteristic clawed hand, or *main en griffe*, is produced.

In cases in which the nerve is completely severed, there is complete loss of muscular response to both faradic and .

galvanic currents when applied to the nerve ; and with galvanism applied to the affected muscles, the 'reaction of degeneration' is obtained.

The **Musculo-spiral Nerve**, owing to its serpentine relations with the humerus, is liable to injury from dislocations and fractures of that bone, or from pressure exerted by a crutch, or from sleeping with the arm twisted under the body.

This nerve supplies directly, or indirectly by its posterior interosseous branch, all the muscles on the back of the upper limb, from shoulder to wrist ; and, in addition, gives cutaneous sensibility (radial branch) to the dorsum of the thumb, and of the index, middle, and half the ring fingers. Consequently, the higher up the situation of the lesion, the more extensive the palsy. If the posterior interosseous nerve alone be affected (rare), the paralysis does not involve the triceps, anconeus, extensor carpi radialis longior, and supinator longus muscles, all of which are supplied by the main trunk of the musculo-spiral nerve before its division. All the other extensor muscles are paralysed.

In a typical complete case there is inability to extend the first row of phalanges, also the wrist, elbow, and shoulder. The second and third rows of phalanges can, however, be partially extended by the unimpaired interossei and lumbricales. Supination is not completely lost, as this action can still be carried on partially by the biceps. Cutaneous anæsthesia involves the dorsal and outer surfaces of the thumb, and the backs of the index, middle, and ring fingers, as far as the ends of the first phalanges. Gowers also draws attention to a prominence on the back of the wrist, which is produced by the bulging of the synovial sacs, and carpal bones which are no longer supported by the extensor tendons.

DISEASES OF THE NERVES

NEURITIS, PERIPHERAL NEURITIS, MULTIPLE NEURITIS

Definition.—Inflammation of nerves in their course or terminal distribution.

Causation.—*Age and Sex.*—Though both sexes suffer from neuritis, it occurs most frequently in women. As regards age, there is no predisposing factor, except in the alcoholic and some other forms, which are found in adults, and the degenerative variety which is seen in old age. *Poisons.*—It is a sequel of chronic alcoholic, lead, and arsenical poisoning. *Cold and Injuries* both act as exciting causes. In traumatism, the neuritis may be caused by direct injury to the nerve; or it may be induced by specific invasion, as in wounds (tetanus). *Specific Fevers.*—Notably diphtheria, small-pox, and typhoid fever. Other specific organisms or their resultant chemical products may also produce the same effects, *e.g.* malaria. *General Diseases.*—It is often a sequel or complication of gout, syphilis, diabetes, rheumatism, leprosy, and tuberculosis. *Locomotor Ataxia.*—The characteristic neuritic pains are one of the earliest signs. *Degenerations.*—Chronic inflammatory changes in the peripheries of nerves are common in senility.

Pathology.—The changes may commence in, and be limited to, the nerve sheath (perineuritis); so that the nerve fibres escape. Or they may begin in the endoneurium (endoneuritis), in which the destruction of nerve fibres is somewhat slow, and is in great measure due to the effects of subsequent sclerosis. Or, finally, they may originate in the nerve tubules themselves (parenchymatous neuritis).

The affected nerve is at first congested, and is therefore red and swollen in appearance ; and when examined microscopically it is found that there is serous exudation, invasion of leucocytes, with proliferation of the connective tissue and nerve-fibre cells. Occasionally small hæmorrhages are seen. The white substance afterwards becomes cloudy and granular, and irregularly interrupted so as to assume a beaded outline. Subsequently the cylinder is entirely destroyed, and is represented only by longitudinal lines of fibrous tissue.

Wallerian Degeneration is a secondary degeneration of nerves bearing, in most respects, a close resemblance to parenchymatous neuritis.

When a nerve, through injury or disease, is cut off from its trophic centre, it degenerates ; the degeneration involving the nerve in the direction of its impulse, but being ' simultaneous throughout the length of the nerve ; ' so that fibrils are equally affected, no matter what part of the degenerated nerve is examined.

The nuclei of the sheath and connective tissue proliferate and interrupt the medullary substance, so that the nerve fibres are broken up and irregular in outline. Then the disorganised fibres become absorbed, and nothing is left beyond the connective tissue and the primitive nerve sheaths.

In some less severe cases a regeneration of fibres may occur. New axis-cylinders bud from the end nearest the trophic centre, and pushing along the track of the old fibres gradually become coated with a new medullary sheath, so that function is restored.

In the case of degenerated motor nerves, the muscles with which they are connected atrophy and lose their tonicity ; their transverse striæ become blurred, or replaced by granular matter. The connective tissue between the fibres is also increased.

Symptoms.—The symptoms vary in their intensity as the disorder is acute or chronic ; they also obviously vary in their distribution, according as the disorder is local or general (multiple neuritis). In any case, however, the onset is, as a rule, gradual.

The symptoms may be concisely grouped according to the physiological functions of the nerves (see Suckling, 'Diagnosis of Diseases of the Nervous System'). Thus, as a result of inflammatory action there is : (1) *irritation of nerve fibres*, causing nerve tenderness (*nervi nervorum*), increased sensibility and tenderness of skin, with cramps, twitchings and deep-seated tenderness of muscles. These signs are characteristic of the early stage. Subsequently there is (2) *cessation of conductivity* of nerves ; hence anæsthesia, paralysis, and loss of deep reflexes. As a result of being shut off from their trophic centres in the grey matter of the cord, the muscles waste, they present the 'reaction of degeneration ;' the skin becomes glossy and hairless, and the nails brittle. Similarly (3) *vasomotor disturbances* are present, such as purpuric patches, and local œdema and perspirations. Bedsores, however, are rare. Mental activity is usually unimpaired, except in alcoholic neuritis, when we should expect delirium and insomnia. There are no bladder nor rectal troubles, unless the nerves to these viscera are also implicated.

As stated above, the extent of the symptoms depends on the distribution of the lesions. The neuritis may be local only, limited to one arm, or leg, or other part ; it may be symmetrical, when both legs or both arms are affected ; or it may be universal or multiple, affecting the nerves of the limbs, trunk, and also, possibly, those parts which are supplied by the cranial and medullary nerves. The legs are, however, the most often affected ; and even if the arms are involved, the paralysis and anæsthesia are more pronounced in the lower extremities. As a result of the muscular paralysis, we find dropped wrist and dropped ankle ; there is complete loss of control over the movements of wrist and ankle joints ; the attachment of the feet to the ankles appears so loose that they can easily be shaken about in that apparent uncertain manner which has been aptly described as 'flail-like.' But, although the muscles are flaccid and the limbs loose, rigidities after a time supervene owing to the action of the unopposed healthy muscles.

Varieties: (1) Alcoholic Paralysis, Alcoholic Paraplegia

This form of neuritis is common in young women who have indulged in alcoholic excesses. It is therefore somewhat prevalent amongst barmaids, disappointed or unhappy wives, and loose women; people who are not so much prone to 'bouts' of drinking, as to secret 'nippings' of spirits or strong wines.

The patient complains of lancinating pains in the lower extremities, and a somewhat ataxic gait, as in *tabes dorsalis*. She has deep-seated pains and tenderness in the muscles, notably of the calf. Occasionally there is hyperæsthesia of the skin, but not always. She also has pains down the courses of the nerve trunks; afterwards followed by anæsthesia and analgesia. The muscles atrophy; they lose their faradic irritability; the knee-jerks and other deep reflexes are absent. In addition, the skin may present purpuric patches, or it becomes glossy and dry on the fingers; and the nails have a tendency to become furrowed and brittle. As a result of her loss of muscular power, the position of the legs when the patient is lying in bed is peculiar. The foot is not flexed, but falls over in the position of plantar flexion (commonly called extension); the ankle joints appear extremely lax, so that on grasping the leg in about its middle third, the whole foot can be easily shaken about in all directions.

Subsequently a similar state of things extends to the forearms and arms, and hence the patient is confined to her bed; she is thoroughly helpless, and has to be assisted in feeding, and in the performance of the necessary functions of life.

In addition—and this is a point of considerable value in diagnosis—she has gastro-hepatic disturbances, and perhaps the mental condition common to people who have largely indulged in alcoholic liquors.

Delusions and hallucinations are also not uncommon.

(2) Post-Diphtheritic Paralysis

The onset of this condition is sudden, about two or three weeks after an attack of diphtheria. The symptoms may commence from the very first appearance of the false membrane,

and are apt to be overlooked. Usually, however, attention is somewhat abruptly drawn to the condition by some difficulty the child has in swallowing, at a period in convalescence when all apparent anxiety is over. Any muscles in the body may be implicated, but those of the palate are generally first affected. The patient has then some difficulty in swallowing solid food ; if he attempts to drink water, it returns through the nostrils. The soft palate is flabby, motionless, with impaired sensation. Consequently, besides the difficulty in swallowing, the patient's voice is 'nasal' and 'grunting.' If the paralysis extend to the muscles of the tongue and lips, there would be difficulty in speech, apart from the pharyngeal intonation. The muscles of the eyes are, however, more frequently involved than those of the face. Different varieties of squints are therefore common ; but internal strabismus is most frequent, attended by diplopia and impaired accommodation. Occasionally the muscles of the neck are palsied, so that the child, when brought into the room, has its chin resting on its sternum ; or the head will remain in a state of extension if the chin be forcibly raised. Moreover, the paralytic condition may extend to the muscles of the jaws, or to the legs, trunk, and arms ; and all the symptoms may be present as are recorded under Multiple Neuritis.

The muscles are wasted, they lose their faradic irritability ; and the knee-jerk is absent.

One or two points, however, require to be emphasised. The affection may involve the heart muscle and cause terribly sudden death. In one case, in which death occurred during defæcation, the cause was probably sudden ventricular paralysis. Again, we have a record of a case in which fatal syncope occurred in a child whilst at play : the nature of a previous sore throat had been overlooked.

The paralyses are usually symmetrical ; they are progressive in character ; they are apt to extend in one region as they improve in another ; nevertheless, it is rare to find all the above forms in one subject, the affection being usually limited to the fauces, or to the eyes, or to the arms in each individual case ; they tend to recovery, and of themselves are not dan-

gerous to life, unless the cardiac muscle, or the muscles connected with deglutition or with respiration, are involved.

The skin may present various anæsthesiæ, or hyperæsthesiæ, either over the region of the paralysed muscles, or in various areas and patches. Neuralgiæ of all forms are not uncommon, and often persist many years. We have seen two cases in which a dragging, aching pain, extending down the arms, coming on after exertion, has been present since diphtheria was contracted many years ago. Tactile sensation is usually blunted.

Lastly, the neuritic symptoms do not necessarily bear any proportion to the severity of the diphtheritic attack. It is as common, or commoner, after a mild as after a severe attack.

Diagnosis.—(1) FROM LOCOMOTOR ATAXIA.—Although an ataxic gait may be present in the early stage of multiple neuritis, there is an absence in the latter disease of the Argyll-Robertson pupil, whilst the muscles show the ‘reaction of degeneration,’ and also are extremely tender.

(2) FROM LANDRY’S PARALYSIS.—This disease is unattended by abnormal electrical reactions. Nor are there any of the remarkable sensory disturbances which are such marked features in multiple neuritis.

(3) FROM MYELITIS.—Here we should find the rapid formation of bedsores, and total loss of control over the sphincters.

(4) FROM CHRONIC GENERAL SPINAL PARALYSIS.—In this disease the progress of the paralysis is characteristic. It begins in the feet, and extends to legs, thighs, and trunk; then similarly, and in order, it involves the muscles of the hands, forearms, and arms. In multiple neuritis, on the other hand, although the muscles of the legs and feet are primarily involved, the disease soon secondarily manifests itself in the extensors of the wrists; the muscles of the trunk and hands remaining, meanwhile, intact.

Prognosis.—Generally favourable; but, of course, much will depend on the cause. In alcoholic neuritis, and other forms due to poisoning, the prognosis is especially favourable if the patient can be removed to healthier surroundings.

Recovery of muscular power also is dependent on the amount of the destruction of tissue.

Treatment.—There are two essentials, viz.: (1) absolute rest in bed for a lengthened period; (2) nourishing food, of a light and easily digested quality, and in quantity which is increased as the appetite grows. In many cases a cure results from this treatment alone, without medicinal aid.

Hot fomentations and anodynes may be applied to the painful limbs, or they may be wrapped in cotton-wool. They should also be supported on splints, to avoid contractures and other deformities.

If the pains are very severe, they may be relieved by opium, or Indian hemp (tinct. cannabis indicæ *mx.*); or by the application of chloroform and belladonna liniments with cotton-wool.

In alcoholic neuritis, absolute and immediate abstinence from alcohol must be enforced. This strict discipline is difficult to maintain in private practice; and if the case shows no signs of improvement, we must suspect that alcohol is somehow or other obtained.

In diphtheritic neuritis, patients usually recover after a prolonged course of iron. Strychnia may be added with advantage. We may also, in all cases, hasten the restoration of muscular power by frictions and well-regulated massage. If, after a reasonable time, the muscles do not improve, resort at once to electricity. The continuous current is best, and it should be applied over as large an area of muscle as possible.

Use such strength of current only as will produce contraction without subsequent pain. Cod-liver oil may be given during convalescence.

NEURALGIA

Definition.—Pain, which occurs in paroxysms, in the course and distribution of a nerve.

The term should strictly be limited to those cases in which the pain is not due to any nerve lesion, but is entirely functional.

Causation.—(a) **PREDISPOSING.**—*Age.*—The disease is rare

before adult life. *Sex.*—Women appear to suffer much more frequently than men. *Heredity.*—The disorder may be inherited as a neuropathic manifestation transmitted from parents, one of whom may have been epileptic, hysterical, or even insane. *Debilitating Illness.*—Any condition or illness by which the general system is reduced, may act as a predisposing cause. Amongst such conditions may be mentioned syphilis, general malnutrition, frequent pregnancies, prolonged lactation, and the ill health produced by anxiety or business worry.

(b) EXCITING causes may be referred usually to one of three large groups, viz. : (1) *central irritation*, such as occurs in cerebral or spinal tumours, and injuries ; (2) *peripheral irritation*, resulting from caries of the teeth, the pressure of a crutch, or of an enlarged abdominal or other organ. It may also be caused by the direct effects of cold, or injury to a nerve. (3) *Blood disorders*, as gout, malarial diseases, relapsing fever, hay fever, lead poisoning, and anæmia. The morphia and alcoholic habits may also be included here as a cause of neuralgia.

Pathology.—In pure neuralgia there is really no pathological change in the nerve. Gowers puts forward the suggestion that there is some disturbance in the nerve-cells with which the central end of the affected nerve is in communication.

Symptoms.—The characteristic symptom is pain in the course of the affected nerve. It is often preceded by tingling or cutaneous numbness. When the pain is once established, it is variously described by different sufferers as lancinating, or crushing, or burning, or grinding ; it supervenes in paroxysms, with intervals of freedom of long or short duration ; and even during the paroxysm itself, there are waves of agony, with intervening lulls, when the anguish is less severe, but which, perhaps, cause more distress than a continuous pain. The distress is often accompanied by vaso-motor disturbances, such as pallor, and arrest or alteration in the characters of various secretions.

There are, in addition, certain painful spots at definite

fixed anatomical points, according to the nerve involved. For the most part, they correspond to the situations of exit of the nerve through a bony foramen, or where it perforates deep fascia.

As the attack passes off, the pains become less severe ; the intervals between the exacerbations are more prolonged ; and the patient generally acquires rest and sleep, but with a sensation of bewilderment and exhaustion. Over the neuralgic area the skin is frequently tender for some days afterwards ; or its tactile sensibility may be diminished or lost ; or a crop of herpetic vesicles in the course of the nerve may indicate the severity of the attack. After frequent recurrences the hair may be blanched, or it may fall out. Usually, the attack is not attended by any increase of temperature.

Neuralgia due to central disease is, as a rule, not a local pain. It is distributed, and affects several nerves, which are often widely separated. In addition, there are symptoms, whether mental, cerebral, or spinal, which mask, or predominate over, the neuralgic distress ; and the pain is often bilateral.

In peripheral neuralgia the patient complains of pains which follow the distribution of one nerve or some of its branches ; there are painful spots here and there in the course of the nerve, which correspond to cutaneous twigs.

In the neuralgia of blood disorders, the pain is often distinctly periodic, and is generally relieved by rest, good food, and quinine.

Varieties.—A *malarial* form of neuralgia is not uncommon. It most frequently implicates the first division of the fifth nerve, causing a ‘brow-ague.’ The principal features of the attacks are, that they supervene only in those who have been the subject of previous ague ; they often commence about ten o’clock in the day ; they are attended by lachrymation, sneezing, loss of appetite, and general discomfort ; the symptoms disappear somewhat abruptly towards evening, and they are relieved quickly by full doses of quinine or arsenic.

But the affection is not limited to the fifth nerve ; the sciatic and others are distinctly liable to malarial neuralgia.

Trigeminal Neuralgia, see Tic Douloureux. .

Cervical Neuralgia.—Neuralgia of the cutaneous branches of the cervical nerves is not uncommon in women. The pain extends upwards to the scalp ; or across the front of the neck ; or, more commonly, downwards to the shoulder, chest, and arm. The disorder may originate in cold, rheumatic affections of the deep fascia of the neck, or enlargement of the cervical glands. Tender points are formed where the nerves become superficial and run over bony eminences.

Brachial neuralgia chiefly implicates the cutaneous branches of the circumflex, intercosto-humeral, and of the ulnar nerves. The patient often complains of sore points in front of or behind the edges of the deltoid (circumflex), over the inner condyloid ridge, and in front of the ulnar side of the wrist (palmar cutaneous).

Intercostal neuralgia is frequently accompanied by an herpetic eruption (zona). The pain is often intense, and there is usually much cutaneous hyperæsthesia. Tender spots occur external to the spinous processes, or in the axillary line, and at the sternum ; over those aræ, in fact, where the cutaneous twigs are distributed.

Similar neuralgic affections may involve the cutaneous branches of the lower six *intercostal* nerves and of the *lumbar plexus* ; so that pain and tenderness occur in the abdominal walls, and in the groin, scrotum, and upper part of the thigh.

Sciatic Neuralgia, see Sciatica (p. 731).

Other varieties are named according to different organs, the nerves of which may be involved. Possibly angina pectoris may be regarded as a form of neuralgia. But certainly the nerves of the uterus, ovaries, mammae, testes, kidneys, and other organs may be affected.

Diagnosis chiefly depends on the intense pain, which is intermittent and paroxysmal. Apart from its purely functional character, the disease may have a local origin, such as occurs in caries and other diseases of bone or of periosteum, the pressure of tumour, the existence of calculi, or other obstructive disease of any canal ; or it may be due to organic disease of the brain, cord, or their membranes. A very chronic pain would point to its being a pure neuralgia, as opposed to

organic disease, which would be expected to develop other signs in addition.

The suggestion that a bilateral neuralgia is due to central disease is not always borne out by clinical facts.

Treatment.—No disorder has had more remedies advocated than neuralgia ; and probably no disorder responds with less regularity to any fixed treatment.

General Treatment consists in removing all possible causes of reflex irritation, if such can be discovered, and in improving the patient's health. Anstie recommended the administration of fats in large quantities, such as cod-liver oil, cream, &c. A change of residence to a warm but not too relaxing climate will often be beneficial.

Internal Treatment.—Amongst the drugs which are of great use are, arsenic, in increasing doses ; bromides with salicylates (especially in rheumatic subjects) ; citrate of caffeine (gr. iij.) ; colchicum (in gouty patients) ; gelsemium ; chloral ; quinine ; phenacetin (gr. v.) ; phosphorus ; hyoscyamus ; morphia ; and alcohol. In anæmic subjects, iron appears to be the most useful remedy.

So far as our experience goes, quinine and croton chloral have been the most uniformly successful. Quinine should be given in large doses (gr. x. to gr. xx. t.d.). The symptoms of quinism appear to be no more severe after the large doses than after the medium ones. Croton chloral may be advantageously combined with gelsemium.¹ Brandy, port wine, or other form of alcohol often relieves, especially when taken with hot water. Morphia had only better be administered when other remedies fail, and should be discontinued immediately after relief is obtained. A similar remark applies to alcohol.

Local.—The liniments of aconite, belladonna, or chloroform are usually of great service. They may be used separately ; but are often better combined. Lint saturated with the liniments should be applied over the painful part, and covered

¹ ℞. Croton-chloral Hydrate gr. iij.; Gelsemin gr. j.; Mucil: Acacie q.s. Ft. pil.

lightly with cotton-wool, so as to allow of evaporation. Blisters applied over the painful spots often afford speedy relief. Menthol, veratria, and opium may also be tried. The subcutaneous injection of hydrochlorate of cocaine (m.j. of a solution gr. iij. ad 3j.) is often useful.

Lastly, the question of nerve-stretching, or of neurotomy, may have to be considered. In some cases surgery has been successful, in others it has been a failure. Even nerve-stretching, therefore, should only be advocated after repeated changes of drugs have failed to procure relief.

SCIATICA

Definition.—Neuralgia of the sciatic nerve, in its trunk, or at its peripheral distribution.

Causation.—Any ætiological classification is most difficult, as many of the causes overlap; and in the following list it will be found possible to group many of the different conditions under two categories, central (cord) and peripheral. There are, however, certain (*a*) **PREDISPOSING FACTORS**, viz.—

Age and Sex.—Most cases found in adults or middle-aged men. *Occupations.*—Those exposed to the effects of cold and injury, *e.g.* 'bus and cab drivers, railway servants, riverside labourers, bargemen, and the like. *Climate and Locality.*—It especially prevails in cold, damp countries; the combination of cold winds, with exposure to wet weather, being potent causal factors.

(*b*) **EXCITING CAUSES.**—*General Diseases.*—It may occur in syphilis, apart from pressure of gumma; it occasionally complicates gout; or it may be a manifestation of malarial disease, notwithstanding that the primary ague may have occurred some years back. It is frequent in rheumatic patients, and also in those who are subject to rheumatoid arthritis, especially when it involves the hip joint. *Effects of Pressure*, either inside the neural canal, or in some part of the course of the sciatic nerve. Thus it may result from caries, or exostosis of vertebrae; syphilitic and other tumours arising from dura mater or other membranes; inflammatory

or other new growths involving the cord itself. Again, it may result from the pressure exerted by aneurysm of the internal iliac artery or of one of its branches; by retro-uterine or other pelvic hæmorrhage; by pelvic abscess; by a loaded rectum. It may be caused by the weight of a pregnant or diseased uterus; it may be consequent on parturition (large fœtal head; forceps delivery).

Finally, it may exist as a simple *neuralgia*; or as a simple *neuritis*. Occasionally, but rarely, it is a complication of diabetes.

Pathology.—In the few cases in which the sciatic nerve has been examined, neuritis has been the principal lesion. It has usually been limited to the sheath and circumference of the nerve, but occasionally found extending to the offshoots of the perineurium; and in severe cases the nerve tubules have been found permanently damaged. The affection is almost always unilateral, unless it be due to organic disease of the spinal cord, or of the cauda equina.

Symptoms.—The symptoms may commence gradually with numbness, tingling, and other discomforts in the course of the nerve, but not of sufficient severity to cause much complaint. Then exacerbations of pain occur, usually sudden and severe, so that it becomes a distressing affection, calling for relief. The patient complains of pain and tenderness, most marked usually at the sciatic notch, but extending down the trunk of the nerve into the ham. So far, the seat of pain is easily localised, demonstrated by the patient's finger; but below the knee, the pain may be limited to parts supplied by one of the great subdivisions of the nerve; or it may be general over the front of the leg, in the calf, round the outer ankle, and on both surfaces of the foot. The character of the pain varies. It may be dull and aching, or sharp, lancinating; or in 'flashes' down the limb, as a patient often describes it.

It is usually increased by walking, or by the pressure of digital examination. The nerve itself and its branches are also tender to the touch; and extremely sensitive points present themselves in the various parts of the nerve where it impinges upon, or is stretched against bony or resistant points,

such as at the great sciatic notch, the middle of the thigh under the biceps, the head of the fibula, the external malleolus, the dorsum of the foot. Occasionally herpetic eruptions are seen. There is no wasting of muscles, or loss of electrical reactions, unless the neuritis is so severe as to shut off the muscles from their trophic nuclei. Nor is anæsthesia a general rule. If it be present in any marked degree in the buttock and upper parts of the ham, it is suggestive of implication of the small sciatic nerve.

The course of the disease is usually a chronic one. Relapses are common; and even when the attack is cured it appears to predispose to future outbreaks. For some time after all acute signs have subsided, there is often a tenderness in the course of the nerve and its distribution, together with a stiffness in the muscles.

Treatment.—Rest is the first essential. Many cases occurring in out-patient practice get well by complete rest in bed for a few weeks. In obstinate cases, a long splint may be applied.

Locally, during the acute stage, apply hot fomentations, or anodyne liniments (opium, belladonna), over the course of the nerve. Aconite ointment is frequently of great service. Blisters are of great advantage, especially if repeatedly applied. In obstinate cases, relief is sometimes afforded by dry-cuppings along the nerve, or by puncturing the nerve with a stout needle at several points of its course. Or we may inject atropin or cocaine subcutaneously. Lastly, the actual cautery, when other remedies have failed, has been often successful.

Medicinally.—Many drugs have their advocates. And in cases where the disease is a pure neuritis, it is a matter of surprise how a given remedy will succeed in one case, and fail in the next. We have found the best results from large doses of quinine (gr. v. to gr. x. t.d.). Cases of rheumatic tendency will be cured by a combination of salicylate of potash with bromides and iodides. Phenacetin (gr. iij. t.d.) is an admirable remedy. We may also resort to caffeine or thein. In anæmic cases, give arsenic.

There is, however, beyond a gouty, syphilitic or other constitutional taint, no guide to our medication. Probably one or two remedies will have to be tried before the efficacious one is hit upon.

It is important to maintain the general health, and to ensure a daily complete evacuation of the bowels.

TIC DOULOUREUX

Definition.—Neuralgia of the fifth cranial nerve, accompanied by spasmodic convulsions of the muscles of the face and jaws.

Causation.—The predisposing causes are much the same as those which are mentioned under Neuralgia. Tic, however, is especially a disease of adult and senile life ; and in many instances an attack has been plainly traced to irritation produced by cold, or by carious teeth. Malaria would also appear to be the starting-point in many instances.

Another factor, as yet not so fully recognised as it ought to be, is the anatomical arrangement by which the fifth nerve, in all its divisions, traverses numerous canals, cavities and foramina. So that it is particularly liable to irritation, either from neuritis in itself, or from changes in the bones and periosteum which produce pressure on the nerve.

Symptoms.—The neuralgic pains involve the distribution of the fifth nerve on one side. They may be limited to one or other of the three divisions, or may implicate all alike.

If the *first* division is affected, the pain extends to the forehead and scalp, the upper eyelid, the eye, and the side and tip of the nose.

In affection of the *second* division, the pain is distributed over the cheek, the lower eyelid, and the upper lip, gums, and teeth.

When the *third* division is attacked, the pain extends to the lower jaw and gums, the tongue, the ear, and the temporal region.

Tender points occur, for the most part, where the nerves escape through foramina, or where they course along the side

of bony eminences. They are therefore found at the supra-orbital notch, at the outer and inner angles of the orbit, at the end of the nasal bone, at the infra-orbital foramen, over the malar bone, at the mental foramen, and over the zygoma.

The pain may, as stated, be limited, during a paroxysm, to one division of the nerve ; but it not infrequently changes to another at a succeeding attack. Each paroxysm lasts from one to three or four minutes ; but they may be very numerous throughout the day. The pain varies from a slight tingling to the most intense agony ; its character being variously described as burning, grinding, or like an electric shock. Movements of the face, a draught of cold air, or mental excitement, frequently originate or intensify the paroxysms. With all this torture, convulsive movements may take place in the muscles of the lips, jaws, and eyelids. The movements, however, are partly emotional ; yet, by their continuation, they may produce permanent wrinkling or distortion of the face. Vaso-motor disturbances also occur, such as salivation, lachrymation, flushes and sweatings of the skin, and the like. The hair of the face and scalp may, in severe cases, turn rapidly grey and fall out ; or be rubbed or pulled out by the constant movements of the patient's hands across the painful part.

Prognosis.—Not, as a rule, favourable, so far as a cure is concerned. The disease usually lasts throughout life ; but the attacks can be considerably modified and postponed by careful treatment.

Treatment.—In addition to the drugs which have been mentioned under Neuralgia, some others appear to be especially beneficial in tic. Croton chloral is extremely useful. Begin with gr. v., and increase the dose up to gr. xv. or xx. Gowers recommends a combination of arsenic, quinine, and Indian hemp. Morphia, however, appears to give the best results. Try the effects of small doses (gr. $\frac{1}{6}$) injected subcutaneously. Occasionally the subcutaneous injection of warm water has equally beneficial results.

Nerve-stretching has been practised, but the good results are apparently temporary only. Division of the nerve is also advocated ; or extirpation of a short piece has had better results. Rose, Ballance, and others have removed the Gasserian ganglion, and apparently with relief to the sufferer.

FUNCTIONAL DISORDERS OF THE NERVOUS SYSTEM

HEADACHE

The causes of headache are many, and extremely diverse in their nature. No classification can be quite satisfactory, as so many of the conditions and diseases which cause headache act through one or two different channels ; and hence a headache which is regarded as due to some vascular derangement, may be caused primarily by some toxic agent. Again, it is a question whether we should regard the headache common in gouty subjects as of vascular causation, or uræmic. Perhaps the most convenient arrangement is to group the different causes under four headings, viz. : (*a*) those forms which are due to diseases external to the cranium ; (*b*) those due to intracranial disease ; (*c*) toxic ; and (*d*) symptomatic or sympathetic.

(*a*) The chief conditions arising external to the cranium are, rheumatic affections involving the fascia and pericranium, syphilitic affections of bones, caries of teeth, and organic diseases or defects of the eye and ear.

(*b*) The intracranial causes are more grave. Headache occurs in meningitis, in cerebral congestion, tumour, and hæmorrhage, and also in cerebral tubercle. It also attends cerebral anæmia, and cerebral exhaustion consequent on prolonged study or mental strain.

(*c*) Amongst toxic causes, we may mention uræmic poisoning, the excessive use of alcohol, opium, tobacco, and other drugs. Uric acid is also another cause of headache ; but whether from its toxic effect, or from the arterial tension with

which its circulation in the blood is associated, does not appear to be clear at present.

(*d*) Headaches of more or less severe form are common in all types of fever. It is difficult to decide whether in these cases the headache is due to the circulation of poisonous matter, or to cerebral congestion, or whether it is merely symptomatic of febrility. Headache also occurs in disorders of the stomach, both structural and functional; it is common in constipation, and functional disturbances of the liver.

The following table will show the chief causes, and their classification, with some of their distinguishing symptoms.

(a) External causes	{	Fascia and pericranium (rheumatism)	{	Often local, with tenderness of scalp	
		Bones (syphilitic disease) . . .		{ Pain intense, with nocturnal exacerbations	
		Teeth (caries)			
		{	Disorders of semicircular canals	{	
			Ear- Suppuration and other affections		
			Caries of petrous bone		
(b) Internal causes	{	Eye: errors of refraction; overstrain, <i>e.g.</i> picture galleries, &c.	{	Referred to brows and eyeballs	
		Meningeal congestion		Relieved by walking	
		Meningitis	{	Attended by double optic neuritis	
		Tumours of meninges, or of brain substance			
		Hæmorrhages			
		(c) Toxic	{	Cerebral anæmia	{
Cerebral exhaustion	Relieved by sleep				
Uræmia	{			Attended by diminished urine	
Alcohol in excess				{	Relieved by abstinence
Opium in excess					
Tobacco in excess					
{	Tea and coffee in excess	{	Attended by arterial tension		
	Uric acid				

(d) Symptomatic	{	Cold			
		Fever			
		Disorders of stomach . . .		{	Attended by foul tongue and other signs of dyspepsia
		Disorders of bowels . . .		{	Relieved by purgation
		Disorders of liver . . .		{	Attended by retching and vomiting
		Disorders of uterus			

Symptoms.—Headache may only be a small, trivial discomfort, which merely predisposes to neglect of occupation ; or, on the other hand, the pain and agony may be so excruciating as to cause symptoms bordering on mania ; it may be local in character as in hysteria, or it may extend all over the cranium as in meningitis ; it may be attended by tenderness in the course of cutaneous nerves ; or it may be relieved by a bandage applied tightly round the head.

That form of headache associated with rheumatic diathesis, is local in its distribution, and is generally attended by some tenderness of scalp, so that the act of brushing the hair causes pain and distress.

The headache due to syphilitic disease of cranial bones is often intense and agonising ; it is worse at night ; and there is usually some tenderness or obvious enlargement of bones.

Headache which accompanies disease of the bones containing the organ of hearing is also most intense ; and if suppuration occur, we should expect to find an irregular temperature curve, and, sooner or later, bulging of the membrana tympani, or discharge of pus by the external auditory canal.

The ocular forms are detected by the ophthalmoscope or by the use of trial lenses, if there is structural deformity of the eye. It also is consequent on constant visual strain, such as occurs after a visit to a picture gallery, or witnessing a cricket match, or after a prolonged journey over snow and ice. This form of pain is usually referred to the eyebrow and forehead ; or to the eyeballs themselves.

The congestive variety supervenes after severe study, anxiety, and other active mental processes. It may also be a

result of heart disease, ehronic bronchitis, and other diseases by which a meehanical congestion is produced. Not infrequently it aecompanies obstinate constipation. It is usually increased by lying down.

Headache due to aetive inflammation of the brain or its meninges, or to cerebral tumour, is attended by double optic neuritis, vomiting, and fever. It is usually severe and constant, and is aggravated by noise and excessive light. The vomiting is generally sudden and urgent, without premonitory nausea.

The toxic headache of uræmia comes on slowly ; it not infrequently ends in delirium, and appears to be relieved by it, as the complaint of pain is not so constant during the lucid intervals. It is usually occipital ; but there is no constant rule. Examination of the urine will reveal albuminuria and diminished urea.

In opium poisoning, the headache supervenes only after reeovery. The pupils are contracted ; the respirations are slower than normal, though not so slow as in the aeute stage ; and the seeretions generally are arrested. Vomiting, however, is not infrequently complained of.

In belladonna poisoning, the headache is also aecompanied by delirium and dilated pupils, with diplopia and other retinal disorders.

Headache consequent on exeessive use of tobacco, tea, or eoffee, is usually frontal, and in many respects resembles that form met with in dyspepsia. Vomiting and nausea, with cold perspirations and pallor, are the usual symptoms.

Febrile headache is usually frontal, but not always.

There is another form of headache, usually situated immediately over the external angular proecess of the frontal bone, which is assoeiated with an east wind or the advent of snow.

Siek headache or migraine is, perhaps, the most distressing form of all, as the patient usually retains his faculties, notwithstanding the distress occasioned by the disease. The pain is often loeal, or limited to one half of the eranium. It is preceeded by double vision or other ocular disturbanees ; or

by deafness ; and is followed by nausea and vomiting, which appear to afford relief. (See Megrin.)

As a general rule, the observations made by Hughlings Jackson are correct, viz. : that headache due to disorders of stomach and bowels is usually frontal ; that due to cerebral lesions is vertical ; and that caused by disorders of circulation is occipital.

Treatment.—Only a few rules can be laid down as to general treatment of headache. Rest is of the first importance in nearly all forms, except the congestive variety. Food should be light and digestible ; and, as a rule, alcohol should be forbidden. Here, however, experience of individual cases is of more value than general rules. In one case (anæmia) headache is relieved by alcoholic stimulant, whilst in another case alcohol aggravates the trouble. The same applies to tea and coffee.

The following remarks refer chiefly to the functional or symptomatic forms of headache. Purgatives are indicated when headache is attended by a thickly coated tongue. The most efficacious are mercury (pil. hydrarg. gr. iv.), podophyllin (gr. $\frac{1}{3}$), and chloride of ammonium (gr. x.). Oxide of zinc (gr. iij.), guarana, thein or caffein, and bismuth are useful in nervous exhaustion.

Brow headache is often instantly relieved by quinine.

In rheumatic conditions, salicin, or the salicylates combined with bromide of ammonium, give speedy relief.

Iodides are necessary in those forms of headache dependent on tertiary syphilitic lesions.

As regards disorders of the ear and eye, their treatment is mainly surgical. In all cases of constant headache occurring in young subjects, the eyes should be carefully examined, and refractive errors, or other ocular malformations, corrected.

Similarly, headache due to uterine or other disorders of the special functions, can only be arrested by appropriate local treatment.

MEGRIM

Definition.—A disease, occurring in paroxysms, attended by headache, disturbances of the organs of special sense, and vomiting.

Causation.—(a) **PREDISPOSING.**—*Age.*—The disease commences usually about puberty, and often continues through life. *Sex.*—Women suffer more frequently than men. *Occupation.*—It affects those who lead an inactive, sedentary life ; or those whose occupation entails much study, mental work, or ocular strain. It is therefore more common in the educated classes. *Heredity* is often a pronounced predisposing feature. It also occurs in those who inherit a neuropathic tendency, such as epilepsy.

(b) **EXCITING.**—The direct exciting cause may be some error of diet, or prolonged mental exertion or anxiety, or some exhausting occupation, whether of pleasure or duty.

Pathology.—No pathological change is known. From the paroxysmal character of the attacks, it bears some resemblance to epilepsy ; and if so, the various phenomena of the disease would be accounted for by periodic accumulation of energy in the cells of the cerebral cortex, which is succeeded by their hyper-physiological discharge. This would, in a measure, coincide with Liveing's and with Fagge's hypotheses, who respectively regard megrim as a nerve storm and a paroxysmal neurosis. The symptoms certainly point to the disease as being cerebral in origin, and not gastro-intestinal.

Symptoms.—Usually there are three stages in an attack.

The *first* is marked by some functional disturbance of vision or hearing. Thus the sufferer may see flashes of light ; or he may have blindness of one half of the field of vision (hemianopia) ; or he may, whilst being blind to surrounding objects, perceive figures floating tremulously across the visual field. These figures are fantastic in shape and colour ; they may represent transverse lines, or rings, often with zigzag or crenated borders (fortification figures), and in iridescent colours. One patient has compared his visual symptoms to the effects produced by forcibly closing the eyelids, or by firm pressure of his finger on the globe of the eye. His hearing may also be affected, though not so frequently. A patient may complain of complete deafness ; or he may be subject to hissings, drummings, or other noises.

Or, again, he may be aphasic ; he may suffer from slight

paresis of an arm, or a leg, or some of the muscles of the eyeball. Another distressing symptom attending this stage is intense mental depression, and a fear of some impending catastrophe.

The *second* stage is characterised by headache. This varies considerably. It may be quite local, or limited to one half of the cranium, and attended by tenderness of the scalp; or it may be general, and so intense as to thoroughly incapacitate the sufferer from any mental or bodily exertion. It also varies in character. In some it is 'boring,' resembling the hysterical clonus; in others it is bursting, or it may be lancinating; but, whatever its type, it is aggravated by noise, by light, and by movement. It is also accompanied by pallor, by coldness of extremities, and local diaphoresis. The pulse is usually small, slow, and feeble. The duration of this stage is uncertain; it may last a few moments only, or persist all day. In some few cases it is the earliest symptom, the primary stage being absent.

The *third* stage succeeds with retching, vomiting, and more or less rapid relief of the more painful symptoms. But the patient remains exhausted and listless; and complains of a sense of weariness, and of tenderness over those parts of the cranium where the pain was located.

The interval before the next attack may vary from a few days to some months; but, as in epilepsy, the patient usually feels quite well, and is able to follow his usual occupation between times.

Prognosis is favourable. Death, perhaps, never occurs as a result of megrim. On the other hand, the patient is liable to recurrences nearly all his life. The attacks, however, may disappear, or be replaced by asthma, epilepsy, gout, or some other paroxysmal disorder.

Diagnosis.—The disease can only be confounded with epilepsy (*petit mal*). But in megrim there is no loss of consciousness whatever; and headache is a marked feature, whilst it is comparatively rarely complained of in epilepsy.

Treatment.—During the paroxysm, the great indication is rest, in a darkened room from which all noise is excluded.

The patient should be made comfortable in bed or on a couch, with hot bottles to his feet and, if desired, cold lotions to the brows. Many drugs have been advocated, with a view to cut short the attack ; but no two cases appear to be identical in their response to any one medicine. Guarana (gr. xv.) appears to afford relief in many instances ; antipyrin (gr. x.) and eaffein (gr. v.) are useful in others. Failing these remedies, we may try the effects of large doses of bromide of ammonium (gr. xx.), or valerianate of zinc (gr. iij.), or ext. cannabis indicæ (gr. $\frac{1}{3}$), or nitro-glycerine (gr. $\frac{1}{150}$), or of amyl nitris (m $\frac{1}{2}$). They appear to act by relieving arterial spasm.

One patient under our care always resorted to an emetic, with a view to hasten the final stage ; and always with success. Purgatives, such as blue pill or podophyllin, are also indicated whenever there is constipation with diminished bile in the evacuations.

During the intervals, the patient should pay strict attention to his diet, abstaining from large meals and indigestible food. He should also avoid, if possible, undue mental excitement and strain, and also prolonged exhausting bodily exertions. But, after all, the patient's own experience will afford the best guide as to what he should avoid, and his own intelligence will lead him to correct errors which induce an attack.

APHASIA

Definition.—Loss of the function of speech. It is usually due to injury or disease of the brain.

The function of speech has its cortical centre in the left hemisphere, except in some left-handed subjects. This suggests, however, the possibility that the cultivation of speech is associated with a similar cultivation of movements of the right arm and hand, and that there is a centre for speech in the right hemisphere, but it is ordinarily in abeyance.

To understand the various forms of aphasia, we must recognise that there are, and that there must be, for the acquirement of speech, certain sensory or perceptive centres beyond those concerned in the special senses of hearing, sight,

touch, and smell or taste (fig. 35). These centres, by which we appreciate the meanings of sounds and forms (word-hearing, word-seeing), in turn transfer or distribute impulses to the motor cortical centres (C), in which reside the *knowledge* of the various movements which are required to speak, or to write, or read. This knowledge is only acquired, and the centres, therefore, only fully developed, by education and training; and hence by presiding over the lower nuclei (N), which control the muscles of articulation or of written thought, intelligent speech

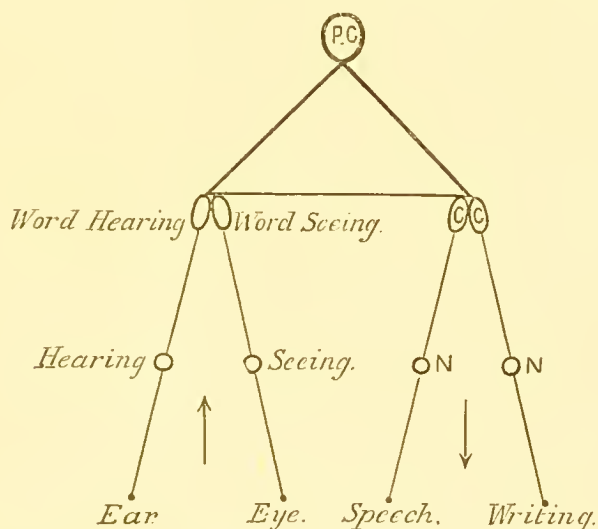


FIG. 35.—DIAGRAM TO SHOW METHOD OF PRODUCTION OF APHASIA

or correct writing is produced. There is a still higher or psychical centre (P C), by which sound, vision, or other stimuli are intelligently appreciated, and by which the value of spoken or written words is properly estimated. This property helps mainly to distinguish man from the brutes. Of course, this highest centre may generate impulses which terminate in spoken or in written thought, independent of the interference of afferent stimuli.

There are, therefore, obviously two forms of aphasia, Sensory and Motor.

In *Sensory* aphasia the mind is blind or deaf, or otherwise

blunted, to quite familiar objects. Thus the sufferer fails to recognise the use of (say) a pen, or a hammer with which he may have been accustomed to work. If one of these objects is given to him, he handles it with no more intelligence than a savage would handle a stethoscope. He appears to read from a perfectly familiar book, but the letters and the words convey no meaning to him. Or he hears ordinary words spoken to him, and they are as a foreign language; a bell is sounded close to him, and he hears it, but it conveys no meaning as to how the noise is produced. Again, in some instances the odour of (say) a pinch of snuff is perceived, but conveys no idea as to its origin. In short, the patient cannot understand what he sees or hears or feels. An object is an object to him, and a sound a sound, but nothing else.

Inability to recognise written words (word-blindness) may exist alone, or with motor aphasia. Usually, however, motor aphasia is present. Thus, though the patient fails to recognise his own printed name, he may be able to write it; but quite as often begins the initial letter correctly and then goes on with a meaningless jumble of pothooks.

The lesion is situated in the angular gyrus and that part immediately surrounding the upper end of the Sylvian fissure.

Inability to comprehend words (word-deafness) is usually associated with motor aphasia. The patient can hear correctly, but cannot understand. Familiar words are as a foreign language. Although the patient can occasionally speak, and gets through a short sentence correctly, he, sooner or later, gets off the track, and jumbles words together in an incongruous or parrot-like utterance: that is to say, with word-deafness there is also, as a rule, some motor aphasia.

The lesion is situated at the upper edge of the first temporo-sphenoidal convolution.

A condition of musical 'tone-deafness' occurs in some people who are perfectly healthy. They cannot distinguish one musical note from another; and yet such people will often attempt to sing, happily unconscious of being distressfully out of tune. The musical tone centre in these subjects is probably not developed.

In *Motor aphasia* the patient cannot remember the efforts which are required for articulate speech ; the word-motor centres are disturbed. He can, therefore, read to himself, but not aloud ; he knows a pen when he sees it, and will demonstrate its use, but he cannot recall its name, although it appears almost on the tip of his tongue. But he will nod acquiescence if a bystander correctly names it ; or, by other gesture, he will show his disapproval if, for experiment, the pen is called a knife. Occasionally, if the word 'pen' is mentioned to him he can repeat it ; but only rarely. Similarly, he cannot write, though he may make a copy of a drawing.

The lesion is in the lower end of the third left frontal (Broca's) convolution.

Few cases, however, are so simple as a brief description would imply. The conditions, in most cases, are exceedingly complex, and require much patience and ingenuity to understand.

In young people the power of speech may be restored by cultivation of the centres in the opposite side of the brain ; but in adults recovery is comparatively rare, and only after tedious education.

The most usual causes of aphasia are, embolism of the middle cerebral artery, hæmorrhage from the same vessel, or tumour.

EPILEPSY (FALLING SICKNESS)

Definition.—A functional disorder of the brain, due to a hyper-physiological discharge of the cells of the cerebral cortex, characterised by convulsive seizures of temporary duration, in which there are clonic and tonic muscular spasms, with unconsciousness.

Causation. (a) **PREDISPOSING CAUSES.**—*Age.*—The onset is most frequent between the tenth and twentieth years. It may, however, occur at any age, although it is rare for a first attack to occur after forty. *Hereditary influence* is often strongly marked, especially in families with neuropathic tendencies, such as dipsomania and hysteria. These factors are more potent in the offspring of consanguineous marriages.

(b) **EXCITING CAUSES.**—Some disturbance of mental functions, such as may be brought about by fright, anxiety, overwork, injury to the head, venereal excesses, and alcohol (especially absinthe).

Pathology.—No fixed lesion has been established. During an attack, the brain is successively anæmic and congested. Post-mortem examinations have revealed the existence of capillary hæmorrhages, especially in corpora striata, pons, medulla, and cerebellum. These changes are probably results rather than causes. In those cases which terminate in insanity, there has been found chronic thickening of the meninges.

The pathology is thus at present very unsatisfactory. It would appear much more likely that the disease is due to want of stability of the cortical cells of the brain; and that at certain periods, and under sufficient provocation, these cells discharge their energy, and produce a 'fit' characteristic in symptoms of the functions of that part of the cerebrum in which the eruption occurs. Thus, if the disturbance be limited to certain circumscribed areas about the fissure of Rolando, the motor convulsions would be limited to leg, arm, or face (see Jacksonian Epilepsy). On the other hand, the explosion, commencing in a motor centre, may extend to those in the neighbourhood, and thence to the rest of the cortical centres, with resultant universal muscular convulsions, loss of consciousness, and other effects. Again, the instability and cortical discharge may be confined to the frontal or other lobes, and hence produce a condition of mental activity known as the 'dreamy state,' an activity which is so marked that no mental process can be fixed or retained. Thoughts and mental observations rush past, as it were, in a confused procession, and leave the sufferer in a state of intellectual stupor. See Hughes Bennett, 'Lectures on Epilepsy' ('B. M. J.,' 1879); also case of 'Intellectual Aura,' by the author ('Lancet,' 1890).

Symptoms.—There is consequently great diversity in the form and the severity of the attack. In a typical case there is a sudden and prolonged loss of consciousness, with muscular

spasms and other symptoms which constitute an attack of *haut mal*.

In other cases the insensibility is temporary, occasionally amounting to giddiness only, accompanied by pallor (*petit mal*).

An attack of *Haut Mal* has been divided into three distinctive stages. *First Stage*.—Characterised by a sudden cry or groan, and a complete loss of consciousness, during which the patient falls to the ground ; his muscles, especially those of the trunk, become slowly contracted and rigid ; the respirations cease ; and the skin is pallid and cold. This stage lasts about thirty seconds. It is occasionally preceded by a well-defined premonitory aura.

In the *second stage* there is a return of respiration ; tonic spasms of the muscles are replaced by clonic spasms, especially in the limbs and face ; the patient is still unconscious ; his face is flushed or livid ; he grinds his teeth, or bites his tongue ; accumulated mucus is churned up in the buccal cavities ; and the pulse is full and throbbing. This stage lasts from about two to four minutes.

The *third stage* is denoted usually by a partial return to sensibility, followed by a rapid and complete recovery. But the patient may remain confused, or become quarrelsome or maniacal. Any way, the muscular spasms cease ; the face is pale and bathed with perspiration ; and his aspect vacant and bewildered. Subsequently he falls into a deep sleep, from which he awakes fatigued, and, it may be, bruised by his fall and struggles, but with his mental faculties restored.

The above is a sketch of any ordinary attack. It is necessary to describe in greater detail some of its phenomena. The prodromal symptoms vary considerably. There is occasionally a well-defined aura, though it is not by any means constant, as we find it, on an average, in only one case in ten. But it is constant in the individual. A patient who has experienced an aura, will probably have one in subsequent attacks. In three members of a family who were epileptic, an aura occurred in each, prior to every seizure. The aura may consist of a peculiar creeping sensation, commencing in the fingers and extending to the head, or beginning at the feet and ‘run-

ning' up to the epigastrium. Or it may consist of disorders of smell or sight, or other subjective sensations. In others, it is marked by depression, or by boisterous laughter, or by vomiting, or by spasmodic twitchings. Whatever form the aura takes, it terminates in unconsciousness, unless (as is possible) the seizure is arrested by pinching the hands, or by rubbings, or other mechanical means. It is interesting, however, to note that the aura makes a deep impression on the memory, as it can always be recalled and described by the patient after his recovery.

In the first stage the features are horribly distorted ; the pupils are either contracted or dilated (usually dilated), but insensible to light. Respiration is stopped, owing to tonic spasm of the respiratory muscles and fixity of the glottis. The pulse is small and frequent, or irregular. A thick mucus and saliva pours from the angles of the mouth.

In the second stage the convulsions may be so strong as to need restraint and protection ; they are usually unilateral at first ; the laboured and hurried breathing, the full pulse, and flushed countenance, the churned-up and bloodstained, or frothy saliva, are due to clonic spasms of the muscles in general. For similar reasons, the contents of the bowels and of the bladder are often discharged in the clothes. The patient still requires restraint, so that he shall not injure himself.

When recovering, he may be stupid or somnolent ; but not infrequently he is obstinate or quarrelsome, often resenting by blows any care or interference on the part of friends.

After a seizure there is copious urination ; and the patient usually complains of headache, probably due to cerebral congestion and perivascular extravasations. In females the attacks are often regularly periodic. The attack is not attended by any elevation of temperature ; it may only last a few minutes, or it may be much more prolonged ; or a series of fits may be included in one seizure, lasting some hours or even days (*status epilepticus*). In this, the temperature may rise as high as 105° or higher, the condition being one of great gravity.

In an attack of *Petit Mal*, all the above symptoms are not observed ; or, indeed, the various stages and phenomena succeed

one another and pass off so quickly as to be unrecognisable. Usually the symptoms consist of a temporary giddiness, during which the patient, if a child, runs to its nurse and catches hold of some support ; he has some mental confusion, slight insensibility, and inability to stand, with pallor and loss of speech.

The whole attack passes away in a few seconds, and appears to consist mainly of a prodromal stage of *haut mal*.

Complications.—BODILY INJURIES caused by falling on the ground, or in the fire, under vehicles, and the like.

MENTAL DERANGEMENTS.—Between the attacks, the patient is liable to sudden frenzies or other forms of madness. He may inflict injuries on himself or on his friends. He may be prone to theft or other moral offences, and occasionally is suicidal or homicidal. Trousseau mentions cases in which the sufferers have performed indecent acts, or have undertaken prolonged walks, or gone through some other performance of which they had no subsequent knowledge whatever. An epileptic patient at St. Thomas's Hospital was allowed to go out one afternoon. He was brought back to the hospital late in the evening, having unconsciously walked to Stratford-by-Bow, when, on recovering his senses, he asked the police where he was, and how far from the hospital. How he avoided the busy traffic of the City will never be known.

Prognosis.—The disease may be cured in early life, if there be no organic disease of the brain. On the other hand, prognosis is unfavourable when the attacks are well-established, or when the disorder supervenes after puberty. Although at first there is usually perfect mental recovery between the attacks, as the disease progresses they become more frequent and severe, and the mind is affected. The patient becomes obstinate ; his memory fails ; and there is usually a progressive and gradual impairment of faculties, until he becomes idiotic or imbecile.

Under suitable care, epileptics may live to a good age ; but the mortality is heavy, not so much on account of the disease, as from accidents and injuries incurred during the attacks. When there is any tendency to idiocy or to mania, or if muscular paralysis supervenes, recovery is hopeless.

Diagnosis.—The attack has to be distinguished from Apoplexy, Hysteria, Eclampsia, and Malingering.

In APOPLEXY, we are guided by the age of the patient (over forty); the longer duration of the fit, which is marked by stertor and snoring; there is also an absence of foam from the mouth, and after recovery the patient has hemiplegia. The sudden fall and convulsion of the primary stage, however, are now admitted to be epileptiform in character.

In HYSTERIA, the paroxysms are attended by greater violence and noise, and they last longer. The patient, moreover, rarely bites her tongue or injures herself; she never fouls her clothing; and the pupil reflex is always present. The sex and age of the patient would be confirmatory evidence.

ECLAMPSIA, occurring in a child, should cause inquiry about teething; or whether the stomach is overloaded with indigestible food; whether there are intestinal worms; or if there has been any exposure to infectious fever.

In an adult there may be evidence of cerebral tumour, of traumatism, of alcoholic or other poisoning, of chronic renal disease, or of parturition. The urine and cardio-vascular system should be examined for evidence of Bright's disease. In most cases the attack is of comparatively short duration, and is preceded by drowsiness; but the symptoms are identical with those of epilepsy, with certain modifications as to severity and duration. Recovery is either quick and perfect; or recurrences take place and destroy life, the patient usually dying comatose.

MALINGERERS, as a rule, overdo the symptoms, especially in the violence of muscular spasms. Search the mouth for soap or other foam-producing article. The pupil reflex is present. Suspicion would be aroused if the would-be patient is a tramp or other social failure, and if his display occurred in a quiet, secluded, and affluent quarter of the town. Recovery is, of course, rapid when the police are summoned, or when a galvanic battery is applied.

Treatment.—(a) DURING AN ATTACK.—Place the patient flat on his back, with the head raised; remove the collar, or

other impediments to free respiration. If possible, place a cork or other suitable padding between the teeth. The patient should also be restrained, so as not to injure himself. No attempt should be made to administer brandy or other restoratives, as there is danger of their passing down the air-passages. A dose of calomel or of croton oil, however, may be placed on the tongue with advantage, in a patient with a constipated habit.

(b) AFTER AN ATTACK.—Hygienic care and precautions are very necessary. The diet should be light and wholesome. Avoid full, heavy meals. Regulate the bowels; order fresh air and moderate exercise; but over-fatigue and excitement and venereal excess should be strongly discountenanced. Although employment involving slight mental exertion is beneficial, the patient should give up all arduous and prolonged mental occupation.

(c) PRIOR TO AN ATTACK.—An attack may be sometimes averted in cases where the premonitory aura is experienced. Various methods have been advocated. Occasionally chloroform or other anæsthetic has been tried with success. Nitrite of amyl inhalation has also been advocated. Various mechanical means may be tried to ward off an impending fit, such as shampooings and rubbings of limbs, and over the epigastrium; or the application of a blister; or a tight ligature or bandage round the limb, above the point in which the aura commences. It is, perhaps, needless to say that all these precautions are only successful in cases which present a definite and prolonged aura.

Certain drugs appear to have prophylactic effects on epilepsy, notably bromides and belladonna. Bromide of ammonium, or of potassium, should be given continuously until bromism is produced. Great relief is obtained by a combination of bromide of sodium (gr. j.), bromide of potassium (gr. ij.), and bromide of ammonium (gr. iij.). These doses may be gradually increased. Even if these remedies do not cure the disorder, they undoubtedly defer the attacks and render them much less frequent.

Belladonna, in some instances, is quite as beneficial. Give

gr. $\frac{1}{5}$ of the extract three times a day, till its full physiological effects are produced. Arsenic, oxide of zinc, nitrate of silver, may also be tried if the bromides and belladonna fail. We have experienced good results from administering oxide of zinc with belladonna.

The applications of setons and blisters to the nape of the neck have been recommended, but their advantage is not very marked.

The treatment of *petit mal* is embraced in that of *haut mal*. A due attention to diet and hygienic surroundings is chiefly indicated.

Jacksonian Epilepsy

Definition.—A condition, first described by Hughlings Jackson, characterised by convulsions limited to one side of the body, or to one limb, or to one set of muscles, due to irritative lesion in the motor area of the cerebral cortex.

Symptoms.—The chief symptoms are a well-marked premonitory aura, followed by convulsions which begin in one limb, and are usually limited thereto (focal symptoms), though they may extend to the rest of the body. There is, however, no loss of consciousness, so long as the discharge of cortical cell energy is limited to the motor area; but should the disturbance extend to other parts of the cortex, insensibility will then occur, though late in the attack.

In other cases the convulsion may begin in, and may be limited to, the muscles of the face, or tongue, or eyeball. The local character of the disturbance is its chief characteristic; although such disturbance may extend in some instances to the greater part or, indeed, to the whole of the body.

Pathology.—In most cases the disease is due to some irritation of the motor cortex, such as that caused by fracture of the skull, or by tumour, or other irritative lesion; and the close observance and order of sequence of the convulsions are thus a good guide to the localisation of the affected area. It is conceivable that occasionally the attack is due to hyperphysiological discharge of the energy of the cortical cells, as in *haut mal*; but such cases, in which the 'storm area' is so circumscribed, are extremely rare. On the other hand,

the effects of a gross lesion may be limited to a definite motor area.

Treatment should be regulated by the general condition of the patient and the frequency of the attacks. A localised new growth holds out hope for relief by surgical operation. For general rules of treatment, see 'Epilepsy.'

ECLAMPSIA (CONVULSIONS)

Definition.—Under this term are included all those many varieties of convulsions which are due to some gross lesion of the brain, or which are produced by toxic matters in the circulation, or which are reflex to some peripheral irritation.

Causation.—**PREDISPOSING CAUSES.**—In all cases there is probably an unduly excitable condition of the nervous system, but especially in those subjects in whom the convulsions are due to reflex irritation. The condition, therefore, is met with more frequently in *women* and *children*, owing to the easier response of their nervous system to irritant stimuli, which would probably produce little or no effect on stronger organisations.

EXCITING CAUSES may be classified under three headings, viz.: (1) *Cerebral Injuries* and *Pathological Changes*, such as depressed fractures of skull, injuries and blows inflicted on the head, cerebral hæmorrhage, the effects of sunstroke, cerebral thrombosis and embolism, cerebral congestions, intracranial tumour (gumma, sarcoma, hydatid cyst), and lastly, cerebral anæmia, or mental strain.

(2) *Blood Poisons*, notably those produced by the retention of waste and effete matter, as in Bright's disease; the effects of specific fevers; alcohol, hydrocyanic acid, and other toxæmics.

(3) *Reflex.*—This form is specially seen in children, as the result of teething, an overloaded stomach, indigestible and irritant food or worms in the intestinal canal. It is also met with in the parturient woman, from pressure of the foetal head on a rigid and unyielding os uteri. Venereal excesses have also been regarded as an exciting cause acting reflexly.

Symptoms.—Generally indistinguishable from epilepsy. The phenomena may be just as transitory as in *petit mal*, or just as severe as in *grand mal*. There are one or two distinguishing features, however. In eclampsia, the ‘fits’ are not so sudden as in epilepsy; there is generally a premonitory condition of drowsiness, headache, vomiting, or restlessness; unconsciousness is not so profound; the attacks are more irregular in their occurrence; they increase in severity as the disease which is their causal factor becomes more chronic, and hence they are more likely to end fatally. They cease, however, when the disease from which they originate is cured.

Further, in eclampsia, one or other of the stages described in epilepsy may be abortive, or, at least, so considerably shortened as to be unrecognised; and the attacks may follow one another so rapidly as to constitute a condition (*status convulsivus*) parallel to *status epilepticus*.

Treatment.—According to the cause and nature of the irritant. The reader is referred to the chapters on syphilis, Bright’s diseases, and others which may be the primary cause of the convulsions.

In children, speedy relief is usually obtained by a hot bath, an enema being administered in the bath. In case of an overloaded stomach or of intestinal hæmorrhage, a powder of calomel (gr. ij.) with ipecacuanha (gr. j.) may be given; it acts as a speedy purge and emetic.

CHOREA (ST. VITUS’S DANCE)

Definition.—A convulsive disease of early life, characterised by purposeless, involuntary, muscular contractions, at first unilateral, then becoming general, but without complete loss of the power of the will. The disease tends to a spontaneous cure.

Causation.—*Age.*—It occurs most frequently between the seventh and fifteenth year; that is, from the commencement of the second dentition to puberty. *Sex.*—Females constitute by far the majority of cases; and especially is this so, when the disease commences in adult life. Chorea in a man

is rare. *Acute Rheumatism*.—Most of our cases have either come of a rheumatic stock, or there has been a decided personal history of rheumatism, either at some period remote from, or immediately preceding, the choreic attack. Occasionally acute rheumatism has supervened during treatment for chorea. *Neuroses*.—Fright, overwork, anxiety, excitement, are all probably predisposing influences. The disorder is occasionally associated with epilepsy. Mimicry, again, has been suggested as a reason for two or more in a family having the disease. We have never yet known cases in which this evidence was quite clear; and it is difficult to admit, in face of the well-known association of chorea with valvular disease, and its probable rheumatic origin, how imitation could act as a predisposing factor. It is more probable that rheumatism acts as a causal element in all the different members of a family which presents several cases of chorea. *Hereditary* influence is often a marked feature, not only as coming through a rheumatic, but also through a neurotic channel. *Parturition*.—The disease is a well-known sequel of childbirth, or a complication of pregnancy.

Pathology.—No constant nervous lesion has been discovered. By some it is thought to be due to *showers of emboli*, in which the corpora striata are especially involved. Against this theory is the fact that the disease is usually unilateral in the early stages. Again, if the lesions were situated in one hemisphere or in one crus cerebri, we should not expect to find the muscles of the eyeball and those supplied by the upper division of the facial nerve to be involved as they are in chorea.

Dickinson describes a hyperæmia of the corpora striata and nerve centres with a general uniform dilatation of the smaller arteries, together with extravasations into their perivascular sheaths; these appearances being most marked in the corpora striata and postero-lateral tracts of the cord. Ord, besides speaking of puddles and extravasations round the smaller arteries, suggests that the nerve-cell processes connected with the fibrils of motor nerves become ruptured, and that the tendency to spontaneous cure which is observed in

chorea is due to the healing of these minute lesions. This theory is at least ingenious and as good as any other. But if there be any pathological change, the inference is that it is not localised in the cord ; since the movements, increased under emotion, are partially controlled by the will, and, as a rule, cease during sleep.

Symptoms.—Probably no premonitory symptoms are observed, and nothing is suspected until the child is seen to have some convulsive movements of the hand, face, or other part of the body. If cases are carefully watched, however, it will be found that there is a period, prior to the typical symptoms, during which the child is fretful and mopish ; or is timid and depressed ; or exhibits a real or apparent mental dulness. The appetite is often irregular and capricious.

Afterwards, the muscular convulsions supervene. It is noticed that the child has a clumsiness on performing delicate movements ; that she cannot sit still in her chair ; and then twitchings are observed, beginning generally in the muscles of the face, shoulder, and one or other hand. The twitchings may be so slight and infrequent as to escape detection. A good plan, however, is to hold the patient's hand as in 'shaking hands,' for some little time, when, sooner or later, a muscular spasm will be felt. The movements are generally one-sided at first ; they then spread to the opposite side, and eventually to the whole body ; although always worse on that side primarily involved.

Occasionally, however, they are limited entirely to one half of the body (hemichorea). When the disease is well marked, the tongue is protruded with difficulty and quickly withdrawn ; the face is contorted with various grimaces ; the muscles of the eyeballs are affected. Often the speech is hesitating and stammering, and deglutition performed with effort.

The movements are in no ways rhythmical, but may be described as jerky (see fig. 29). They consist of sudden impulsive and purposeless convulsions, followed by a period of rest, which is again succeeded by movements in another distinct set of muscles. The jactitations, therefore, are not persistent ; and although described as 'purposeless,' they, in a measure, resemble voluntary movements. They are aggra-

vated by excitement or observation, and by any attempt at muscular co-ordination, such as picking up a pen, or the like ; and then they constitute a mixture of regular and irregular movements. They cease under the influence of sleep or anæsthetics, and may be partially controlled by a strong effort of will. These constant movements produce a typical condition of the patient's bed and bed-linen. The clothes and sheets are speedily dirtied and crumpled, so that the bed is untidy, and the patient looks unkempt, in spite of the most careful nursing.

Rigidities do not occur ; but the muscles show more or less paresis, especially when the affection is unilateral. The convulsive movements may be replaced by hemi- or by para-plegia.

The symptoms, however, are not limited to the muscular system. Sensation is impaired in the majority of cases. Intelligence is blunted during the attack ; hence the child acquires a silly or semi-idiotic expression. She is emotional, capricious, or gloomy. The appetite fails ; the bowels are confined ; but there is no fever.

The disease is liable to remissions, exacerbations, and relapses.

In all cases a cardiac murmur should be expected. The murmur is not functional, but is due to organic disease. The mitral valve is most frequently affected ; regurgitation or stenosis may occur, the former especially, as in acute rheumatism. But any valve, and also the pericardium, may be involved ; and from our record of a large series of cases, valvular disease supervening on chorea is as frequent as in acute rheumatism. The conclusion which is forced on our mind is that chorea is one of the many manifestations of rheumatism occurring in young subjects. The murmur is in no sense hæmic ; nor can it be regarded as due to irregular choreic movements of the heart muscle.

After a time, varying from a few days to some weeks, the convulsions become less severe ; muscular co-ordination is more under control ; the child becomes more intelligent, and is finally apparently cured, except, of course, the endo- or pericardial lesions, if they have occurred.

On the other hand, the convulsions may increase in frequency and in degree; the patient can with difficulty be kept in bed; she loses control over the sphincters; fissures form on the tongue and lips, from frequent nips which are given to them by the teeth; sores form on the prominent parts of the hips, and also about the elbows, not only from pressure, but also resulting from friction during the severe movements. In such cases, death may occur from exhaustion, or from failure of an already enfeebled heart. Delirium is usually present at the last stage.

It is necessary to mention that choreic movements may be entirely limited to certain muscles of the face, neck, arm, or leg (partial chorea), producing unsightly grimaces or contortions. They are usually the result of some trick, habit, or irritation, and often last through life.

Diagnosis.—The typical jerky movements, together with the absence of fever, of tonic muscular spasm, and of coma, will distinguish chorea from any other disease of the brain or spinal cord.

Prognosis.—As a rule, favourable in youth, except the case be very severe; or complicated by bedsores, or loss of control over the sphincters. The tendency to relapse, however, should always be borne in mind. In adults, the prognosis is by no means so good.

Treatment.—Prolonged rest in bed is best, the majority of cases tending to spontaneous cure. Time is on our side. All the so called nerve-tonics have been advocated by different physicians, and as the convulsions almost invariably cease during treatment, each drug has been credited with the cure. Arsenic appears to give the steadiest and most constant good results. Failing this, we may prescribe sulphate or valerianate of zinc, also iron, phosphorus, strychnia, or cannabis indica. Sulphate of eserine (gr. $\frac{1}{20}$) injected subcutaneously has afforded good results. In those cases which occasionally present a high temperature, together with distinct evidence of past or present rheumatism, the salicylate of soda (gr. v.), together with bromides of ammonia or potash, produce speedy relief.

Occasionally amelioration is quickly afforded by repeated

shower-baths, or by the application of ether spray to the nape of the neck, accompanied by massage. In very severe cases, it may be necessary to control the convulsive movements by chloroform, chloral, or opium.

Good diet, fresh air, and hygienic surroundings contribute largely to hasten the cure. The child should, if possible, be isolated from its companions.

PARALYSIS AGITANS

Definition.—A disease of advanced life, characterised by tremors of muscles of the limbs, independent of voluntary actions ; but usually sparing the muscles of the head and neck, and followed by muscular weakness and rigidities.

Causation.—*Age.*—The disease nearly always commences between forty and sixty-five years of age ; seldom before or after. *Sex.*—There is a slight preponderance amongst men ; but it is not very great. *Heredity* has no predisposing influence. *Neuroses.*—It has supervened on mental shock, and other emotions, such as grief or terror. The disease has also, in some cases, been connected with injuries to the peripheral endings of nerves, and also with prolonged exposure to wet and cold.

Pathology.—No definite lesion has been established. Pigmentation of the cells in the grey horns of the spinal cord has been detected in some instances. In others, sclerotic and degenerative changes have been found in the arteries of the brain and cord ; but these are probably only senile alterations. If there is any lesion of the central nervous system, the symptoms would rather point to the grey matter of the cerebral cortex for its situation.

Symptoms.—The onset is insidious. The patient usually complains of some tremor in one thumb or the corresponding great toe, which, gradually increasing, attacks the muscles of the hand or foot, and ultimately extends to those muscles which are situated higher up in the limbs. But in whichever limb the tremors commence, they are at first unilateral in their distribution, as in chorea ; and subsequently extend across to the muscles of the opposite side.

The tremors are rhythmical in character (see fig. 29). If the hands are involved, there is a quick series of movements of the fingers over the pulp of the thumb end, producing an action similar to that of pill-rolling. When the muscles of the forearm are attacked, there are quick movements of alternate flexion and extension of the wrist or elbow.

As the disease advances, the movements increase in rapidity and coarseness so far as the limbs are concerned ; whilst similar, but less exaggerated, tremors are apt to supervene in the muscles of the head, jaws, and tongue. The facial muscles, however, usually escape.

In the later stages, rigidities occur. The head is flexed ; and with a fixed, staring, anxious face, there is thus produced an expression of sadness which is almost characteristic.

The hands also become clenched, and the forearms flexed ; the toes are pointed, the heels being drawn up by the rigid calf muscles.

The patient's gait is also peculiar. If pushed forward when he commences to walk, his pace immediately increases ; and, with feet shuffling along the ground, the knees bent, and the body bowed, he breaks into a kind of jog-trot, as though attempting to overtake his own centre of gravity ; and thus he progresses, perhaps the whole length of a hospital ward, or until he is arrested by a wall, or a piece of furniture, or, it may be, until he falls sprawling on the floor. This is known as *festination*. Or, if pushed backwards, he cannot stop himself, and would probably fall, unless arrested by some fixed object. This is called *retrogression*.

During the progress of the disease, the patient does not lose control over his sphincters ; and the electrical reactions of the muscles, and the tendon reflexes, are normal.

The above is a short description of the principal symptoms of the disease. Some further details are, however, necessary.

The tremors come on in paroxysms, often whilst the patient is quiet and unexcited ; they persist during rest, but may be arrested by a vigorous effort of the will, or by some muscular effort, such as lifting a heavy weight. They may be

limited to the muscles of the hands or of the legs only ; and are liable to remissions and exacerbations, though usually they cease during sleep and under the influence of an anæsthetic. They also, by their persistence, cause exhaustion and perspiration ; or give rise to a sense of discomfort and uneasiness, which is attended by some slight feverishness. Nystagmus is absent.

Rigidity, although usually a late symptom, may often precede the tremors. When it attacks the muscles of the legs, any form of distortion may be caused ; but a condition resembling talipes equinus is most frequent. Owing to the rigidity principally, but in part due to the tremors, the patient walks with a shuffling, scraping gait, and can only turn round with difficulty.

Neuralgic pains in the legs and joints are common at one or other period of the disease, but especially in the early stages, or before tremors commence.

Prognosis.—Generally unfavourable. The disease is progressive, but slow. It is not in itself directly fatal ; but the patient usually sinks from asthenia, accompanied by bedsores and impairment of mental faculties. Pneumonia, or some undercurrent acute inflammatory condition, frequently is the immediate cause of death.

Diagnosis.—The disease may resemble *Disseminated sclerosis*. But it is distinguished by the tremors persisting during rest, and being more rapid and regular than in sclerosis (see diagram 29). Further, there are no nystagmus, and no ‘scanning’ speech ; and vertigo, a marked symptom of disseminated sclerosis, is uncommon in paralysis agitans.

Treatment.—All we can do is to ameliorate the symptoms. Rest is very important. We may also try to improve the muscular tone by shower baths, electricity, and tonics.

No drug has been found capable of arresting the disease. All the so-called nervine tonics have been prescribed, such as nitrate of silver, arsenic, strychnia, zinc. Sedatives appear to afford most relief, especially hyoseyannus, belladonna, and opium, but they effect no permanent improvement. A combination of arsenic and extract of Calabar bean appears to

have given the best results. Ergot of rye also, in some cases, is useful.¹

TETANUS

Definition.—A disease attended by tonic spasms of the muscles, of a painful character, usually commencing in the masseters ; and liable to severe paroxysms.

Causation.—(a) **PREDISPOSING CAUSES.**—*Age.*—It occurs at all ages. *Sex.*—Both equally liable, except in the traumatic form, when men preponderate. *Climate and Race.*—The disease is most frequent in hot countries ; negroes are specially liable.

(b) **EXCITING CAUSES.**—Two great varieties of the disease are described, *Traumatic* and *Idiopathic*. Traumatic tetanus has, for the most part, a surgical interest, as it is apt to occur after wounds and operations however trivial ; and it would appear that a *specific bacillus*, described as ‘bristle-shaped,’ enters the system at the seat of injury. Hence it not only occurs after surface wounds, but may supervene on parturition ; or it may attack the newly-born child, apparently by inoculation through the funis. Idiopathic tetanus appears to come on after exposure to *wet* and *cold*, like rheumatism. But it is difficult to understand how these cases arise, if tetanus is due to a specific bacillus, unless we conclude that the micro-organism can be taken into the system by other channels than an injured surface.

Pathology.—No definite or constant naked-eye morbid anatomy has been described. The lungs have been found to be congested. A similar condition has been described as occurring in the spinal cord and brain ; but all these conditions can be accounted for by the fever and paroxysms of spasm. Other observers have described a shrinking of some of the cells of the anterior cornual grey matter ; but the appearances are not definite.

As regards the wound itself, it has not necessarily any other than a healthy appearance ; but Rosenbach has discovered

¹ R̄. Ext: Cannabis Indicæ gr. $\frac{1}{2}$; Acidi Arseniosi gr. $\frac{1}{20}$; Ergotin gr. j. In formâ pilulæ.

this bristle-shaped bacillus in the tissues, especially in the vicinity of the wound.

Symptoms.—The chief symptoms of disease are (i.) tonic spasms of muscles beginning in one set and gradually extending to all the rest ; (ii.) paroxysms or exacerbations of spasm, in which one group of muscles appears to be especially involved, and followed by intervals of rigidity.

In the idiopathic form, the onset is quick. The muscles which close the jaw are, in the great majority of cases, first involved. The masseters become stiff and rigid (trismus) ; the patient cannot open his mouth ; he cannot chew his food ; and he may have difficulty in swallowing. Pain and stiffness then extend to the muscles of the neck, and so on to those of the limbs and trunk ; also to the diaphragm, and other muscles concerned in respiration.

Paroxysms or spasmodic exacerbations occur from time to time, the frequency of their recurrence varying in different cases. They are excited by noise, or by draughts of cold, or by movements. During the paroxysm the limbs become rigid and extended or abducted ; if the back muscles are affected, the patient rests on his head and his heels, which appear to act as buttresses to his arched back (opisthotonos) ; or when the abdominal muscles are involved, he may be curled round with his head and knees together (emprosthotonos) ; rarely the spasm is concentrated in such muscles as cause lateral curvature of the body (pleurosthotonos). The spasmodic muscles are also affected with cramp-like pains of severe intensity. The patient also complains of dyspnœa and pain on breathing. His dyspnœa is due to rigidity of the diaphragm and intercostals ; whilst any urgent attempt at respiration is attended by fresh access of diaphragmatic spasm and pain.

In the intervals between the paroxysms, the limbs and body still retain their rigidity, which even increases in severity. The face at once expresses great mental anxiety and bodily anguish. The eyelids are often screwed up, as though the patient were making an effort to bear up against the pain ; or at another period the eyeballs are fixed and staring, as in dyspnœa. The forehead is vertically and longitudinally

wrinkled. The mouth is widened and its angles elevated, so as to form an ugly grin (*risus sardonius*). The pulse is quick and feeble. The temperature is only slightly febrile (101° about), but often rises to 103° or even to 109° before death. The patient does not lose control over his sphincters, but the urine is often retained. The bowels are constipated. He sleeps badly, and is often awakened by a recurrence of paroxysms of muscular spasm. Consciousness is never lost; and the patient is painfully aware of his precarious condition almost up to the last moments of life. Sensation also is unaffected.

Diagnosis.—FROM STRYCHNIA POISONING.—In this condition the onset is much more rapid, and its duration much shorter, death occurring in a few hours. The paroxysms strongly resemble those of tetanus, but there is a *complete* remission between, during which the muscles relax, and are not rigid, even if painful. Lastly, the muscular spasms begin in the hands, and not in the jaws, although uneasiness in the masseters is, perhaps, the first symptom to be noted.

FROM HYSTERIA.—This disease, although occasionally attended by opisthotonos, is free from pain; the convulsive attacks are more violent and varied, and are accompanied by sobbing, or laughter, or by some boisterous manifestations.

FROM HYDROPHOBIA.—Here there is a history of dog-bite. The paroxysms affect the muscles of the pharynx. They are excited by attempts at drinking. Delirium or mental aberration is a usual symptom.

Prognosis.—Unfavourable. The traumatic form is especially fatal. Milder idiopathic cases may recover. Most patients die in the first week. The longer the survival, the better the prognosis. Favourable signs to be looked for are, less severity of the tonic spasms, diminution in the frequency of the paroxysms, and gradual fall in the febrile symptoms.

Death is usually caused either by fatal spasm of the muscles of respiration, including those of the larynx, as well as the diaphragm; or by exhaustion.

Treatment.—The patient must be placed in bed in a quiet

and darkened room. All unnecessary movements, both on the part of attendants and of patient, should be avoided. If necessary, give a purge at the onset, and then leave the bowels alone. Food should be given in concentrated form, either by nasal tube, if the jaws be rigid ; or by enemata.

As to drugs, there is a large array to select from, and authorities differ as to the value of each. Our own limited experience is in favour of morphia by the mouth. Hypodermic injections are contra-indicated. Belladonna in full doses may be tried, if the congestion of the central nervous system is a causal lesion. Chloroform inhalations can do no harm and give relief. Other drugs which have good reputations in tetanus are, ext. physostigmatis (gr. $\frac{1}{2}$ every two hours), hydrate of chloral (gr. xxx.), curara, cannabis indica (℥xx.). Whatever drug be decided upon, it must be given in such doses as to produce its full and early physiological effects.

TETANY

Definition.—A disease characterised by tonic spasms of the muscles of the hands and feet, occurring in paroxysms, and, at times, extending to the muscles of the trunk and face.

Causation.—*Age.*—The majority of cases occur between puberty and the twenty-sixth year ; but it may occur in infants and in old people. *Other Diseases.*—It is in some way, not yet explained, associated with certain diseases, either during their activity, or as a result of the exhaustion they occasion. Thus it occurs in rickets, diarrhoea, rheumatism, and chronic poisoning by ergot ; it may complicate convalescence from some specific fever ; it supervenes on over-lactation ; it may be present at one or other period of a pregnancy. *Imitation.*—It is said to spread amongst children by imitation.

Pathology.—No pathological change has been detected in the nervous centres, or in the muscles. Little or nothing is yet known. From a review of its symptoms, it would seem as though there were some irritative stimulation of, and discharge from, the cells of the anterior cornua in the cord.

Symptoms.—As a rule, the disease is sudden in its onset.

The spasms begin usually in the hand and forearm muscles. The wrist is flexed, and also the first phalanges; the other phalanges being extended. Spasm occurs also in the thenar and the hypothenar eminences, so that the thumbs are adducted to the middle of the palm and generally flexed, and consequently the hands are cone-shaped; this action being especially produced by contraction of the interossei, lumbricales and the muscles of the thumb and little finger. In the feet, the ankle is also flexed and pointed downwards, and the toes curled up. The expression of the feet is, however, not so marked as that of the hands (carpo-pedal contractions). As the disease becomes more marked, the elbows are flexed, and the arms adducted. This is in accordance with the law that the flexors and adductors are collectively stronger than extensors and abductors. Occasionally, spasms extend to the muscles of the trunk, causing opisthotonos, or to those of the eyeballs (squints).

After a time, variable from a few minutes to an hour or two, the spasms relax and are succeeded by an interval of relaxation, which is again followed by a return of the paroxysms, usually induced by some stimulation, such as irritation of a nerve, or of the muscles themselves.

The above is a short sketch of the characteristic attacks, but some further details are necessary.

The chief characteristic of the muscles themselves is their excitability and ready response to electrical stimuli. Anodic closure contraction (ACC) is easily obtained, which is a somewhat rare occurrence. The spasms are easily induced by pressure or irritation applied to the muscles themselves, or to the trunk nerves going to supply them. There is not always an interval between the paroxysms; the muscles may relax, but they do not invariably assume their normal healthy tone; in children, indeed, there is often a continuous but less severe spasm. The paroxysms then may be said to *remit*, but not to *intermit*. The contractions in themselves are not usually painful, but pain may be afforded by forcibly trying to overcome the spasms. During the spasms, however, painful cramps may occur. The spasms are not influenced by sleep.

The intervals between the attacks vary from a few hours to some weeks.

During the course of the attack, there is slight febrility, and also some slight vaso-motor disturbances producing sweats, and an almost characteristic local œdema on the backs of the hands and feet.

Prognosis.—The patients recover, as a rule. The disease, however, tends to recur after apparent perfect recovery. Death, if it should occur, is due sometimes to diarrhœa, with which the disease originated; in other cases the fatal termination appears to be caused by exhaustion, or during a convulsion.

Diagnosis.—FROM TETANUS.—The spasms in this disease begin in the muscles of the jaws, and there is no complete intermission between the attacks.

FROM HYSTERIA.—Here the spasms are occasionally unilateral; they are not accompanied by pain, and can be easily overcome when the patient's attention is diverted.

Treatment.—Check the diarrhœa, and treat any supposed causal disease or condition, such as rickets or prolonged lactation, on general grounds. The diet should be simple, nutritious, and consist mainly of liquids.

Bromide of potassium appears to be the best remedy to arrest the disease. Other drugs with good reputations are, Calabar bean, cannabis indica, and also chloroform inhalations.

Electricity (constant current) has also been advocated; but, in view of the excessive excitability of the muscular system, it must not be used without extreme care.

HYSTERIA

Definition.—A nervous disease characterised by convulsive seizures, and numerous functional disorders of the mind and body. The patient apparently suffers from defective volition. Wilks has described it as 'a paralysis of the will.' The patient's expression, 'I cannot help it,' is theoretically a definition of the disorder, which may perhaps be regarded as a form of insanity.

Causation.—(a) PREDISPOSING CAUSES.—*Sex.*—The great

majority of cases are females ; it is rarely seen in men. *Age*.—It is most frequent from the advent of puberty to the twenty-fifth year of age. It may, however, supervene earlier or later. *Heredity* is often strongly marked, either as a direct transmission, or as representing some neuropathic disorder in the parents. *Condition of Life*.—The disorder is far more frequent in single women belonging to the idle or luxuriant classes. It is comparatively rare in married women, except they be childless.

(b) **EXCITING CAUSE**.—So far as the paroxysms are concerned, mental strain and excitement are the principal exciting causes, *e.g.* jealousy, fright, joy, and prolonged mental exertion. But in all cases it is probable that none of these causes act alone, and that they must be combined with failing health and debility. Hence diseases of the ovaries, and uterine disorders, have been regarded as exciting causes. It is more probable that they act as such, only by lowering the general bodily health in those women who have an unusually excitable nervous system.

Pathology.—No pathological changes are known beyond those which supervene on chronic functional disturbances.

The probability appears to be that hysteria is due to some disturbance in the higher centres of the brain, a disturbance which does not necessarily produce microscopical changes even, but by which the will, intellect, and the emotional or other attributes are affected.

Symptoms.—The symptoms may be grouped into two distinct categories, viz. : (a) *Paroxysmal* and (b) *Simulative*, of certain functional disorders.

(a) The paroxysmal symptoms are principally convulsive. The convulsions rarely come on without warning. The patient has premonitory symptoms, such as a sense of uneasiness or tightness at the chest ; a boring pain in the forehead (clavus) ; or a sensation as of a ball rising from the epigastrium or iliac regions to the throat (globus). Occasionally an attack is preceded by neglect of duties ; or by some strange action ; or by an alteration of temperament altogether foreign to her previous disposition. The actual paroxysm

itself, however, is sudden. She falls, or, rather slides, to the ground, or, preferably, on to some comfortable couch or chair ; and is apparently convulsed. At the same time she is noisy with shouts or screams. She is not entirely unconscious, but can be aroused by a voice of authority or by a cold douche. She kicks and struggles ; the muscles throughout the body generally are convulsed, or at least it is very unusual for the spasm to be limited to one limb or part. She possesses unusual strength ; she is difficult to hold : first one set of muscles acting, then another, producing wild and varied movements. She has an altered expression of face ; but her features are never grossly distorted, as in epilepsy. Further, spasms are always clonic, or, at least, rarely tonic ; she does not injure herself, nor does she foul her clothes ; the pupils are usually dilated, but always respond to light ; and the respirations continue all through the attack, although quick and noisy.

The duration of the seizure is irregular, sometimes lasting a few minutes, in other cases some hours ; much depending on the treatment and surroundings. After a time the patient becomes less violent and noisy, and gradually recovers, with, perhaps, immoderate laughter or sobbing. Then she falls asleep ; and after an hour's quiet slumber, she awakes, feeling usually better than ever. The attack is not accompanied by fever, but is succeeded by a copious polyuria. It may be the chief or only manifestation of the disorder, the health between times being good.

(b) The simulative symptoms are extremely wide and varied. They may represent diseases of almost every organ and disturbance of every function of the body. There is no order in their appearance, and no especial frequency of one set of symptoms over any other. It will, therefore, be convenient to group the symptoms on an anatomical basis.

(1) *Nervous System*.—Hyperaesthesia is a frequent symptom. It may be general ; or it may be local, affecting a limb, or a mamma, or an ovarian region ; or, again, it may involve one lateral half of the body, or the lower extremities only. At times it is confined to the abdominal walls, the symptom

being so marked as to simulate acute peritonitis. Frontal headache, and pains in the left side under the mamma are, perhaps, more frequent than any other form. Nor is this hyperæsthesia limited to cutaneous nerves. Some patients are acutely sensitive to noise, light, and smells; in others, joint pains are excessive, and are increased by movements or pressure.

Similarly, anæsthesia affects different parts. It frequently involves one half of the body (hemianæsthesia), terminating abruptly at the middle line. Or it may be limited to the course of a single nerve, cranial or spinal, or to irregular zones and patches corresponding to no anatomical distribution of nerve or nerves. In some instances the anæsthesia is so profound as to allow of severe mechanical irritation or injury; in others it is less marked. As a rule, however, it is more intense than in cerebral hemianæsthesia; and the surface temperature of the affected side is reduced.

Motor paralysis may also affect any part of the body, and to any extent. As in the affections of sensation, it may involve a lateral half of the body, or be limited to the legs or to one limb; but the muscles of expression appear to escape. Paraplegia is most common. Joints may become rigid with tonic flexion. The muscles do not waste, although they become rigid; and there are no bedsores or other signs of trophic lesion; nor is there loss of control over the sphincters.

It is said that qualitative electrical changes are not seen; but certainly, after a time, degenerative changes do occur in the helpless muscles; and, eventually, sclerotic changes occur in the cord. Occasionally an hysterical patient will complain of tenderness in various regions of the spine, accompanied by occipital headache, or pains extending to the arms, round the waist, or down the legs, according to the situation of the spinal sensitiveness. And as this distribution of pain corresponds with the anatomical course of the nerves issuing from the spine at the seat of pain, it would seem probable that there is, in such cases, a pathological change in the cord or its membranes which we have as yet failed to recognise.

In some instances the patient may be mentally in default.

She exaggerates her symptoms ; she causes high thermometrical observations to be made ; she is untruthful, or thievish. She may be dull and apathetic ; or, on the other hand, boisterous and wild in spirits. She resorts to all kinds of mechanical irritations and even tortures in order to excite pity and solicitude ; and is not infrequently the subject of marked delusions.

(2) *Gastro-intestinal Canal*.—Here there is the widest extent of symptoms. Some have imaginary dysphagia ; another complains of epigastric pain and tenderness. Eructations, flatulence, rumblings in stomach, constipation or diarrhœa, or even constant vomiting, may all be the subjects of loud complaint. Indigestion accompanied by spasms and abdominal distension are, perhaps, the most frequent. But with all these conditions, nutrition does not, as a rule, suffer ; the patients often appear plump and rosy. Exception, however, must be made to those cases in which food is absolutely refused, and which may eventually succumb from intense debility and emaciation (*anorexia nervosa*). Also, it must be remembered that hysterical people are always able to live and thrive on a very scanty diet ; and their appetites are not only small, but depraved ; so that they often surreptitiously devour coal, slate-pencil, or disgusting and soiled food.

(3) *Respiratory System*.—Hysterical aphonia, in all degrees, is common. The voice fails most when it is most required. Under other conditions the patient can scream or shout loud enough. A peculiar harsh cough, or bark, is common. In others, the respirations may suddenly be accelerated from a normal rate to sixty or seventy per minute, but without any lividity or other signs of dyspnœa. Laryngoscopic and stethoscopic examinations fail to discover any lesions.

(4) *Circulatory System*.—Under this heading we may notice tumultuous action of the heart, with violent and distressing pulsations in the carotids and other trunk vessels ; local hyperæmia or ischæmia ; or œdema in parts, apparently not dependent on any obvious lesion.

(5) *Genito-urinary Tract*.—There may be retention of urine and also suppression. It is important to recognise the

differences between the two. After a paroxysm there is a large excretion of urine, which may be voided in due course. In others, it is retained, either as a result of paralysis of the bladder; or, more frequently, probably as an inducement to vaginal examination and catheterisation.

On the other hand, the excretion of urine may be diminished, and the amount of urea lessened. This condition is frequently associated with urgent vomiting or refusal of food, two circumstances which may account for the want of kidney secretion.

Amongst affections of the genital organs may be enumerated amenorrhœa, menorrhagia, and other disturbances of the menstrual function; also hyperæsthesia of the vulva, and nymphomania, often associated with indecency or onanism.

In addition to the above, there is a type of hysteria in which the convulsive phenomena are much more severe and varied; the condition being known as *hysteria major*, or *hystero-epilepsy*.

The attack is generally induced by some great emotional disturbance; and when well-marked, there is rigidity of the trunk and limbs, alternating with the wildest movements, the arms and legs being thrown about with great force, and the head violently moved from side to side.

The onset of such seizures is sudden, and is marked by loss of consciousness, followed by tonic, and subsequently by clonic, muscular spasms.

To this succeeds the stage of co-ordinated spasm, in which there is opisthotonos; or in which the limbs are rigidly extended, or the arms stiffly abducted, in the 'cruciform' attitude of Chareot; or in which there are various complex and violent movements and contortions of the body generally. Respiration is irregular and stertorous.

Finally, the third or emotional stage supervenes, which is characterised by wild talking, hallucinations, and the assumption of attitudes or expressions of joy, anger, fear, or indecency.

This variety is almost always attended by tenderness in the ovarian region, pressure over which organs may arrest, or, on the other hand, induce an attack.

Diagnosis.—The hysterical paroxysm itself is not difficult to recognise. Still, it may be confounded with (1) *Epilepsy*. The principal points of difference are that in hysteria the onset is expected or less sudden ; there is some obvious exciting cause ; the patient never injures herself when she falls ; she is never quite insensible ; she never fouls her clothing ; the pupils act to light ; and, finally, the paroxysm itself is longer, and more disorderly and noisy.

(2) *Inflammation of Brain and its Meninges*.—At times the diagnosis is most difficult, many of the symptoms of hysteria being also met with in meningitis. In meningitis we find a high temperature (104° or more), optic neuritis, and loss of pupil reflex. In addition, we should not expect to see so varied, diverse and exaggerated a series of symptoms as in hysteria.

(3) *Other Lesions of the Central Nervous System*.—Cerebral hemianæsthesia is not, as a rule, so profound as in hysteria ; nor does it affect the cranial sensory nerves ; also on account of vaso-motor paralysis, the temperature of the affected side is higher.

Abdominal rigidity and tenderness, or indeed any form of tenderness, may be overcome by attracting the patient's interest to some other symptom, during which palpation may be practised without complaint.

Muscular rigidity of hysteria is an early symptom, and is never followed by qualitative electrical changes ; but it occurs late in cerebral or spinal cord lesions. In the hemiplegic rigidity of hysteria, both leg and arm are equally affected ; if it were due to cerebral disease, the arm should be involved first and most severely. Further, a rigid limb in hysteria may be easily flexed if the patient's attention be diverted to some other channel.

The diagnosis of hysterical aphonia is usually at once set at rest by galvanism.

In other localised symptoms in which the diagnosis is

doubtful from the likeness they may bear to organic disease, we must be guided by the aspect and surroundings of the patient, by the irregularity, and inconsequent and inconsistent character of the various phenomena produced. The patients complain of symptoms of which we may almost say 'They cannot happen ;' and, in addition, we shall sooner or later be enlightened by some boisterous laughter, or by aphonia, or by some act approaching immodesty, which will immediately reveal the true nature of the complaint. In addition, we find nervous symptoms come on suddenly, and as suddenly disappear, a condition which can only occur in hysteria.

Treatment.—(a) DURING THE PAROXYSM.—The patient should be placed on a couch or bed. Loosen garments and all impediments to free respiration. Gentle yet firm restraint may be necessary, so as to prevent the patient doing any injury. The vigorous application of a wet towel or a cold douche to the face and neck will often cut short the paroxysm. So far as our observation goes, such treatment acts by inducing a deep series of inspirations, so that the circulatory functions are restored. Inhalations of ammonia have a similar effect. The idea that an hysterical attack is due to wilful viciousness, is not only wrong, it is cruel ; yet firmness of manner by medical attendant and friends is essential. Treatment which tends to coarseness or severity should be avoided, notwithstanding its advocacy by some authors.

(b) The HYSTERICAL CONDITION requires more general treatment. The first indication consists of obedience to the rules of health. A proper, simple, and digestible dietary should be enforced. The patient should keep regular hours, and should avoid heated rooms, and all tendencies to dissipation or unhealthy excitement. The bowels and other emunctories must be carefully regulated. Exercise and healthy amusements are very essential. Beyond this we should treat the mental condition. Gain the patient's confidence ; it is wrong to sneer at, or make light of her ailments. We should impress upon her the necessity of carrying out our orders, and that she can be cured by her own resolution to obey.

Paralytic affections are usually quickly cured by galvanism or faradic current. The application of various metals (Burg's treatment) to a paralysed or an anæsthetic limb is, at times followed by astonishing results. Some physicians have made light of such treatment, and have suggested that discs of leather or potato would produce equally good results. Possibly so. We can, however, only speak of our own experience. We have seen various forms of motor and sensory paralysis cured in a few hours from the application, in some cases of gold, and in others of silver coins to the affected limb or part. Not only is anæsthesia cured, but the motor power has also been restored. Nor has the relief been affected at the expense of a similar paralysis transferred to other parts. How this procedure acts, is at present beyond our knowledge. Probably some strong mental impulse is stimulated by the gold and silver coins, since a bangle or other article of jewelry will produce a like effect. The influence of fright, or of some prospective joyous meeting, or of witnessing some exciting procession or spectacle, will sometimes cure paralysed limbs and vision, the stimulus acting probably by the same channel as Burg's metal discs.

One of the most difficult conditions to treat, is emaciation due to the obstinate refusal of food. The Weir-Mitchell treatment succeeds best. The patient should be kept isolated and quiet. She should have one or, at most, two attendants. She should be allowed perfect rest, bodily and mentally. The palsied and wasted muscles should be treated daily by massage or by electricity. Food should be given at fixed and regular intervals; the amount of food being small at first, and gradually increased. After a few weeks, the mental condition returns to a healthy state, the muscles develop, and the general appearance of the body becomes plump and well-nourished.

As regards medicines, valerian, assafoetida, and other offensive-smelling drugs have been advocated. Beyond their antispasmodic effects, we doubt if they afford any real benefit in the hysterical state. We much prefer the exhibition of tonics, such as iron, quinine, and arsenic. Bromides, again, have been advocated. They are valuable when an impending

paroxysm is feared ; but continued for any length of time, they do more harm than good, owing to the pustular eruption which may follow, this acting as a factor in continuing the hysterical condition.

Treat vomiting, constipation, and any other functional disorder on general principles. Gowers strongly advocates the subcutaneous injection of apomorphia (gr. $\frac{1}{12}$) for the relief of the paroxysmal spasms.

CATALEPSY

This condition often occurs in hysterical subjects, and, as such, is symptomatic of the hysterical condition.

But it may supervene in patients who have never suffered from hysteria, although there is always a history of some neurosis.

The exciting cause of the attack is generally some severe fright, or religious fervour, or other emotional disturbance. It may also be induced in hysterical subjects by a sudden noise, a bright light, or by persistently gazing at some fixed object.

The chief symptom appears to be loss of voluntary movement, with some retention of muscular power. If an arm, for example, be raised by an attendant, the patient at first resists, then gradually yields, so that the limb can be placed in any position, in which it remains for some time, and then gradually falls to the couch by gravity. This condition, first of rigidity, then of yielding, suggests the appearance as though the limbs were made of wax—hence the term *flexibilitas cerea*.

Other symptoms which accompany the above are, slow breathing, a weakened action of the heart, and, occasionally, complete anæsthesia and loss of reflex movements. The last symptoms, however, are by no means constant, and may even be reversed.

Treatment.—Gowers recommends that, during the attack, attempts should be made to rouse the patient by external stimulation, such as cold douches, snuff, &c. Faradism is

especially useful. It should be applied to a limb or the back, a gentle current being used at first.

During the intervals between the attacks, the treatment is that of hysteria. (See Gowers, 'Diseases of Nervous System.')

NEURASTHENIA

Definition.—Defective nerve strength.

This condition occurs most frequently in men, especially those who follow active mental employments.

It is characterised by a want of nervous energy, owing to which there is, besides other symptoms, an inability to engage in any work or occupation. The patient also complains of want of sleep; his eyesight fails; the pulse and respirations are feeble, consequently he suffers from cold feet, chilblains and other symptoms of defective circulation; and his breathing is shallow or sighing. He has no appetite, and consequently loses flesh. He is liable to excessive flushings of the face, or to bodily sweats; but other secretions are usually scanty, and hence there is constipation. Tender points are complained of in the spine, or scalp, or along the gums; and various neuralgiæ are frequent.

In one case the sufferer has a morbid fear when walking in a crowded street, or has an insuperable dread of being in narrow closed places (claustrophobia). Hence he cannot travel by rail; he dreads to enter a lift; and avoids even a narrow, walled passage.

In another instance the patient fears that he may commit some act, or express some thoughts, which would cause him irretrievable disgrace. Or he dreads, though engaged, to enter matrimony, owing to some fancied defect of sexual power.

Other cases present many of the various symptoms and mental conditions which are associated with confirmed hypochondriasis or hysteria.

There is no actual structural disease, yet the condition is one of great distress, and of some gravity. Although the patient tries to overcome his weakness, and does not appeal

for sympathy, but rather hides his troubles, he may, after a time, become so morbidly depressed and miserable as to require restraint. Cases even present symptoms of confirmed insanity, with a suicidal tendency.

Treatment.—Absolute and prolonged mental rest is the only useful treatment. A long ship-voyage is best. No drugs are of the least avail; and narcotics must be strictly forbidden. Beyond this, a good or generous dietary and amusing occupations are requisite.

The patient should be firmly convinced that he will recover, and he will then probably do so.

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